VOL. 31–B, No. 3 AUGUST 1949

The Journal of
Bone and Joint Surgery
BRITISH VOLUME

INTERMITTENT CLAUDICATION

The association between lameness and arterial disease was recognised over a century ago and yet it is only within the past twenty years, since Lewis explained the pain of claudication, and sympathectomy was practised for its relief, that interest in the subject has grown sufficiently to make claudication in the calf a commonplace. Even to-day, similar pain in the foot or buttock is mistaken too often for evidence of foot-strain or arthritis.

Fuller understanding of muscle pain due to ischaemia might have been expected to lead naturally to rational therapy, but it has been general experience that the results of many forms of treatment which from time to time have been fashionable seem to be unpredictable. The reason for this must be that our understanding is still far from complete, and the publication in this number of the Journal of investigations by Professor Boyd and his colleagues, which make a substantial contribution to our knowledge of this complex subject, is most welcome.

It is important in the first place to correct the usual teaching that there are but two forms of obliterative arteritis—Buerger's disease and arteriosclerosis. Though it is generally true that the pathology of arterial obstruction in youth is different from that in older people, it appears that the degenerative changes that have long been regarded as characteristic of the period after forty years of age may also be encountered much earlier in life. Injury to main arteries, especially to the popliteal artery at the level of the knee joint, is an etiological factor that has not been given the prominence it deserves.

It is now many years since Professor Boyd laid stress upon the importance, in regard both to symptomatology and prognosis, of the level in the arterial tree at which obstruction has occurred. The opportunity he has had in Manchester of observing large numbers of patients with claudication has served to amplify his earlier ideas and enable him to separate his cases into well-defined groups according to the nature and site of the arterial obstruction. As we should expect, the prognosis of the disease, and the response to treatment, have varied considerably in the different groups. Of equal importance, however, particularly in forecasting and assessing the results of different forms of treatment, is the severity of the claudication. Patients who are able to continue walking slowly after the onset of moderate pain, and in whom the discomfort then disappears or persists only as a tolerable ache, naturally do better than those who cannot bear the pain and are quite unable to continue walking until it passes off, thus being forced to stop after a comparatively short distance.

The suggestion, which used to be so popular, that arterial spasm plays a prominent part even in obliterative arteritis becomes steadily less acceptable as more facts about the anatomy and pathology of the circulation are brought to light. Though the occurrence of spasm in large arteries cannot be doubted, it would appear that changes in the peripheral pulses during and after exercise, and certainly the benefits of sympathectomy for obliterative arteritis, can be explained without postulating the presence or relief of arterial spasm.

The decision to advise sympathectomy for claudication is often difficult, involving not only assessment of the condition of the vessels in the affected limb but also, as Boyd points

out, full consideration of the state of the heart, the blood and the lungs, the blood pressure, the metabolism and the general nutrition. It is here that the advice of a medical colleague is so valuable, and this is surely a field in which the best interests of the patient are served only by close collaboration of physician and surgeon.

When sympathectomy is not indicated it is important to know what palliative measures are available, and the Neurovascular Clinic in Manchester is providing the means by which to assess the value of drugs and such surgical procedures as neurectomy and tenotomy. Much has already been done, and in days to come we will look with confidence to the same source for further enlightenment.

J. PATERSON ROSS.

FRACTURES OF THE SPINE

A most stimulating article in this number of the Journal, on fractures of the dorso-lumbar spine, presents strong evidence in support of the thesis that functional results are seldom related to anatomical results and that often the best treatment is to ignore displacement, resist the temptation to immobilise the spine in plaster, and teach active exercises from the beginning. It is suggested that reduction of displacement by hyperextension is not always possible—that when it is possible it cannot always be maintained—that when it can be maintained it makes little difference to the end-result—and that when it does make a difference to the end-result it is to the disadvantage of the patient. With typical cogency and conviction Nicoll claims that the treatment of vertebral fractures by hyperextension and immobilisation in plaster is needless in some fractures, useless in others, and harmful in nearly all.

Some of us were taught as students that patients with spinal fractures should be put to bed between sandbags and protected from every movement until a posterior support had been fitted. Permanent disability was accepted as inevitable. With such training it took courage to believe that complete recovery might be possible, and it was with triumph that we learned to reduce deformity by hyperextension, immobilise the spine in a plaster jacket, and institute early active exercise for the spinal musculature. We believed that an important advance had been made—and perhaps it had. But it is right that triumph should be short-lived; and to-day we are told that it was not the manipulative reduction or the immobilisation in plaster that mattered—the significant advance was the early institution of active exercise. We are urged to believe that many fractures of the spine are best treated by exercise alone.

Nicoll's case is based on careful and detailed analysis of 166 fractures of the spine in miners, whose conditions of work are such that it must be acknowledged at once that any man who continues full work at the coal face for five years after injury has indeed made a good functional recovery. Twenty-four miners made such recovery despite gross anatomical deformity; and the severity of deformity that is illustrated in these pages, and shown to be compatible with full work as an underground ripper, will no doubt open the eyes of many readers. Does this imply that gross deformity is the ideal end-result of the treatment of fractures? On the contrary it indicates that many psychological as well as physical factors are concerned in the determination of function—a truth that is emphasised in the article on traumatic paraplegia by Guttmann, who shows that even agonizing pain of causalgic type after spinal injury is often relieved more successfully by the psychological measures of rehabilitation than by the physical measures of operative surgery. To what extent, therefore, were Nicoll's results independent of the physical standards of his treatment and much more dependent on the excellent rehabilitation service that is available to the miners of Great Britain? Is it possible, for instance, that rehabilitation was applied more successfully when no plaster jacket was used than when it was, thus giving an erroneous impression that the plaster itself was harmful? Willingness to return to work depends no less on the art than

on the science of medicine and we are left wondering whether return to work at the coal face is really to be accepted as a precise scientific measure of physical recovery—a measure upon which many of Nicoll's conclusions are based.

If return to full duty is indeed to be accepted as an estimate of the success of treatment we might quote figures from another service in which rehabilitation was developed to a very high standard but in which nearly every patient with fracture of the spine was also treated by reduction of the displacement and immobilisation in plaster.* In one group of 1058 vertebral fractures, treated by many orthopaedic surgeons in the Royal Air Force, no less than 91·4 per cent. returned to duty, including not only those who engaged in heroic action against the enemy but also those who accepted the daily toil of heavy engineering and other arduous ground duties. Only 6·6 per cent. were invalided. It may be that these results could be improved upon—but not by very much. It may be that some of the treatment was unnecessary; but it would be difficult to concede that any of it was harmful.

The claim that it is unnecessary to reduce minor wedge compression of fractured vertebral bodies may possibly be right. Certainly we learned during the recent war that in simple compression fractures of the upper dorsal vertebrae (which occurred with particular frequency in the Air Forces owing to the use of Sutton shoulder harness which raised the fulcrum of movement from the lumbar to the upper dorsal level) such reduction was impossible and good functional recovery could often, though not always, be gained by early hyperextension exercise. It may be that reduction of minor compression, which is impossible in dorsal fractures, is unnecessary in lumbar fractures—though this writer must admit that in the mobile lumbar region he would prefer not to accept as a very obvious potential source of pain the distortion or, at least, strain of interarticular joints that must surely be inevitable if compression of vertebral bodies is left unreduced.

But it is one thing to claim that reduction is unnecessary and another to claim that it is harmful—as Nicoll does. We are reminded of the dangers of oedema and spreading ecchymosis in muscle planes (as if this did not occur in every fracture), and of the ill-effects of muscle shortening due to immobilisation (as if this was not easily corrected by exercise). It is suggested by statistics and theories that in patients with dorso-lumbar fractures the persistence of lumbo-sacral pain after long-sitting is attributable to immobilisation in plaster, and on this is based the conclusion that immobilisation is harmful. But one young pilot fractured his twelfth dorsal vertebra in a crash-landing in Belgium and within a few hours began a thousand-mile trek along the roads and ditches of France, and the mountain passes of the Pyrenees, before arriving four months later in England with one complaint alone—namely, lumbo-sacral pain after long-sitting. We should hesitate before accepting this symptom as a complication of immobilisation in plaster!

The urge to abandon immobilisation in favour of early functional activity has recently been applied also to fractures of the calcaneus, the tibial tuberosity, the carpal scaphoid bone and the neck of the humerus. And yet many of us are still seeing gross deformity of the foot necessitating late operative treatment which could have been prevented by early manipulation and plaster; instability of the knee joint from lack of early immobilisation of torn ligaments; non-union of fractures of the scaphoid bone which was preventable; and even non-union and mal-union of the neck of the humerus attributable solely to enthusiasm for the historic and out-of-date teaching of Lucas Championniere. Such treatment could still be defended if there was conflict between the demands of anatomical replacement and the demands of functional activity—but there is no such conflict. It is easy to maintain the tone of muscles, and to preserve the movement of joints, despite immobilisation in plaster for three or four months. No visitor to Royal Air Force Rehabilitation Centres during the recent war would have doubted that the tone and volume of his own spinal musculature was vastly inferior

* WATSON-JONES, R. (1947): Paper presented in summary to the Brussels meeting of the International Orthopaedic Society.

to that of pilots whose fractured spines had been immobilised in plaster. The test of success was that the spine was better after treatment than before injury.

It would be unjust to imply that the main thesis of Nicoll's contribution is blind advocacy of early functional activity. On the contrary he has made an important advance, first in establishing that the fulcrum of spinal movement is not, as so often has been assumed, at the level of the interarticular joints, but at the nucleus pulposus; and then in distinguishing clearly the stable fractures that can safely be treated by conservative methods (exercise alone if he is to be believed, and hyperextension in plaster with exercise if we are to be believed) from unstable fractures in which we all agree that either spontaneous anterior fusion or surgical posterior fusion of the intervertebral joint is needed. He has considered in detail the sources of such instability—comminution of the vertebral body, rupture of the intervertebral disc, subluxation of the interarticular joints, and rupture of the interspinous ligament. It may not be right to compare interspinous ligament ruptures with ruptures of the medial collateral ligament of the knee joint, which share with the corresponding ligament of the elbow joint the unusual feature of curling and rolling of the fibres, or even interposition within the joint, so that early operative repair is often required. But, in general, Nicoll's differentiation of stable and unstable fractures is to be accepted.

The important question to be determined is whether or not there is truth in the suggestion that in unstable fractures, spontaneous anterior fusion, achieved laboriously after many months, is better than surgical posterior fusion which can be controlled with precision. We must not of course compare spontaneous anterior fusion of a single intervertebral joint with surgical posterior fusion of many intervertebral joints by the antiquated technique of Albee grafting. Some may read with surprise that " undamaged vertebrae are usually included in surgical fusion so that four instead of two become fixed." Several years have elapsed since we learned to use radiographic control in the operating theatre (taking radiographs after the introduction of a metal marker into one spinous process) so that surgical fusion was limited strictly to one intervertebral joint, or sometimes to the two intervertebral joints that were damaged.

This contribution on the problems of treatment of dorso-lumbar fractures has the very great merit that it is stimulating and provocative of further thought. It establishes the need for differentiation of stable from unstable fractures; it leaves us to consider whether stable fractures are best treated by functional activity alone, or by the protection of a hyperextension plaster jacket together with functional activity; and it opens the question as to whether the best result is gained in unstable fractures from spontaneous anterior fusion, or from surgical posterior fusion of the damaged intervertebral joint.

As to the management and rehabilitation of patients with traumatic paraplegia, on which we have invited in this number of the Journal a contribution from Ludwig Guttmann, whose work at Stoke Mandeville has achieved such success, we would quote only the words of Nicoll from the reports of a group of surgeons who are trying to achieve in Great Britain a general and unified paraplegia service, comparable in efficiency with the magnificent paraplegic units of Canada and the United States of America which have proved beyond doubt the possibility of restoring the victims of paraplegia to happy and useful lives:

" There can be no middle course with this condition. Either it must be treated superbly well or as badly as possible. The worst thing that can happen to these patients, and it is happening, is that they should be treated well enough to prevent them from dying but not well enough to make life worth living." EDITOR.

INTERMITTENT CLAUDICATION

A Clinical Study

A. M. Boyd, A. Hall Ratcliffe, R. P. Jepson and G. W. H. James,
Manchester, England

From the University Department of Surgery, Manchester Royal Infirmary

Lameness in horses during exercise was recognised by veterinary surgeons in France and Germany about the middle of the nineteenth century and it was assumed that in some cases this was due to occlusion of the main arteries of the limb. In 1831, the syndrome was named " intermittent claudication " by the veterinarian Bouley. In 1846, Benjamin Brodie described a similar syndrome in a man " whose femoral artery was converted into a gristly cord, so as to be quite impervious from the origin of the profunda to the point at which it perforates the tendon of the great head of the triceps adductor muscle." He realised that lameness was due to the insufficiency of a reduced arterial supply for the added demands of muscle exercise, and claimed that the condition was to be found principally in " those who drink too much fermented liquor and do not take sufficient exercise " and " especially those who are overfed with animal food."

The description by Charcot (1858) of a patient with an aneurism of the common iliac artery was the first accurate and comprehensive survey of the clinical picture. Charcot (1887) and Marinesco (1896) added further clinico-pathological studies. Erb (1898) collected many examples; he reviewed the literature and emphasized that, when there was arterial disease, lameness or limping during exercise was due to pain and not to muscle weakness. Since that time many excellent studies of the symptomatology and etiology have been published.

THEORIES OF THE MECHANISM OF THE PAIN-SYNDROME IN VASCULAR DISEASE

1) **Muscle spasm**—Earlier clinicians were much impressed by the observation in patients with intermittent claudication that occasionally, after exercise, there was palpable change in the consistency of the calf muscles. They allowed that the primary cause of pain was arterial deficiency, but thought that the final mechanism initiating the pain was muscle spasm. This spasm was likened by Charcot (1858) and Marinesco (1896) to " cadaveric " rigidity, even although the pain of intermittent claudication is continuous and does not fluctuate with each muscle contraction. Erb (1898) gave support to this view; but it was abandoned by later writers because in the light of further investigations it proved obviously inadequate.

2) **Arterial spasm**—To estimate the rôle played by arteriospasm in intermittent claudication it must first be ascertained whether or not there is actually spasm in the vessels of the limb at the time that pain is felt. Evidence has been marshalled by various authors.

During exercise the arteriosclerotic foot pales. Pallor precedes the pain and it has been assumed that it is due to arterial spasm of the skin vessels, and that the muscle arteries might behave similarly, thus causing pain (Thomas 1922, Zak 1921). We too have observed this pallor during exercise but it is not always present. Moreover, pallor may be induced in a severely arteriosclerotic limb by briskly rubbing the skin over the tibia, or by passively flexing the ankle joint a few times. The deduction that muscle vessels and skin vessels behave in similar manner is not justified; nor is the assumption that pallor is due to arterial spasm (*vide infra*).

Lian (*cit.* Thomas 1929) believed that he had demonstrated spasm in the vessels of the lower limb of a patient with arteriosclerotic claudication by arteriography before and after

exercise. Similar studies were repeated in greater detail by Veal and McFetridge (1936) and they showed that exercise-pain was in fact accompanied by dilatation of the vessels, especially those of smaller calibre.

After exercise of the arteriosclerotic limb, arterial pulsations at the level of the ankle joint are diminished, or they may disappear, and oscillometric readings taken on the lower leg are correspondingly decreased (Comroe 1923, Leary and Allen 1941). This observation was confirmed in great detail by Ejrup (1948). Both oscillometry and the palpation of vessels measure pulsation of the arterial wall and not flow of blood. A more reasonable explanation than that of arterial spasm is that inflow through the major vessels of the limb is limited by the arteriosclerotic process and that these vessels are unable to dilate normally in response to the demands of exercise. When structural changes are less advanced, the arterio-capillary bed of the muscles increases greatly in capacity and in the course of exercise it abstracts a much larger share of the normal blood supply than when the limb is at rest; and moreover, since the systolic stroke of the major vessels is distributed to a greater bed than before, pulsation in the medium-sized vessels which is measured by oscillometry is decreased.

Claudication has been described in limbs which in the resting state showed no arterial abnormality; yet, on exercise, there was characteristic pain and the pulses disappeared (Pearl 1937). Simmons (1936) stated that: "A small proportion of cases of intermittent claudication are due to pure spasm of the arteries in neuropathic individuals and no structural disease of the vessels is demonstrable either at the time or in later years." Such exceptional cases can be explained more satisfactorily by postulating a thrombotic lesion proximal to the femoral artery. The collateral circulation is good but not sufficient to supply the great needs of exercise. It is noteworthy that the only case of "spastic claudication" described in the literature that has been reviewed at a later date was proved to have organic constriction at the lower part of the aorta. (Lindquist 1948, *cit.* Ejrup.) Two of the authors of this article (Boyd and Jepson 1949) have seen two cases of thrombosis of the external iliac artery with a similar syndrome.

3) **Muscle ischaemia as a cause of pain**—In a series of analytical papers, Lewis and his co-workers demonstrated that intermittent claudication was due to inadequacy of blood flow through muscles during exercise causing accumulation in the tissue spaces of a pain factor—"factor P"—which stimulates the sensory nerve endings (Lewis, Pickering and Rothschild 1929; Lewis 1942). Katz (1935) suggested that factor P was a non-volatile acid. Lewis showed by occlusion-plethysmography that the arterial bed was dilated at the time of the onset of pain.

An extension of Lewis's conception of muscle ischaemia causing pain—In the normal limb it is the muscles that are worked most strenuously in relation to their bulk that first give rise to pain. On the other hand, in limbs suffering from arterial disease, instead of all muscles having a blood supply that is approximately equal in relation to their bulk, the arteriosclerotic process alters the distributive pattern of the arterial supply either by general narrowing of the arteries or by local thrombosis. This redistribution differs from limb to limb according to the anatomical structure and the extent and topography of the arteriosclerotic lesions. It is the arterial inaccessibility of certain parts of the muscles that causes impaired nutrition and accounts for pain on exercise. Pain then arises in that muscle, or part of a muscle, that is worked most strenuously during the complex pattern of walking, and in which the arterial supply relative to the demands of exercise is most deficient. This pain on exercise disappears only when the collateral supply becomes adequate, or when the arteriosclerotic process increases and the affected muscle undergoes atrophy, or is replaced by fibrous tissue so that its potential as a focus of deep pain is destroyed.

In the arteriosclerotic limb, careful palpation of the musculature reveals patches that are tender to firm pressure. This tenderness is, of course, elicited only in superficial muscles and it is noted with particular frequency in the gastrocnemius and sometimes in the small

muscles of the sole of the foot such as the extensor digitorum brevis or in the dorsi-flexors of the foot. It may be obvious only after recent exercise, especially if this has been sufficient to give rise to the pain of claudication. The area of muscle tenderness varies. In limbs that are severely compromised by arterial insufficiency a single muscle or muscle group may be distinctly tender; but in better nourished limbs the patch often measures only a few centimetres in diameter. A site that is often tender is the upper part of the medial belly of gastrocnemius.

The pain suffered on pressure is likened by the patient to the pain of intermittent claudication. The cause of tenderness is not certain but it is probably metabolic in origin and depends upon the same factor that accounts for the pain of exercise. When pain is induced by exercising muscles under ischaemic conditions the accompanying muscle tenderness, and the pain itself, disappear rapidly when the blood flow is re-established although as Lewis (1942) pointed out "a little tenderness may remain for an hour or more if the test has been repeated several times." In arteriosclerotic limbs the involved muscles are continually on

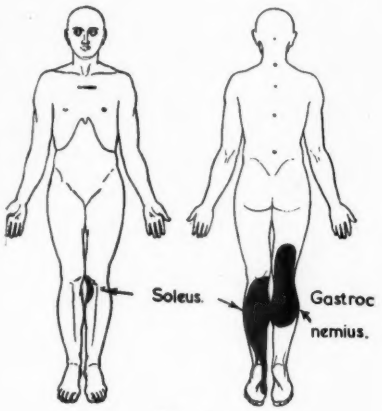


FIG. 1

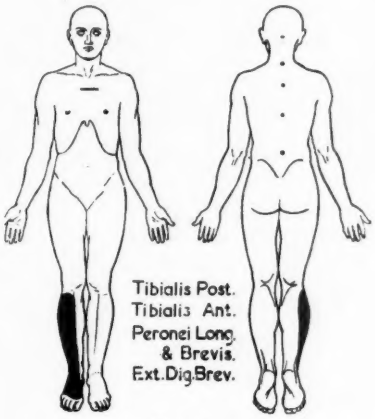


FIG. 2

Segmental reference of deep pain from the gastrocnemius and soleus (Fig. 1) and from the anterior tibial and peroneal muscles (Fig. 2).

the threshold of ischaemic pain and thus the tenderness, which may never entirely disappear, is always emphasized by exercise. More intelligent patients may notice a patch of deep tenderness while kneading the calf muscles, but usually no such story is elicited because surface representation of the deep pain may be distant from, and not overlying, its place of origin.

Infiltration of the tender area with a few cubic centimetres of 2 per cent. novocain makes it anaesthetic to deep pressure and, by blocking the deep pain trigger-point, it allows the patient to walk painlessly for a distance far exceeding that which was possible before injection. The increase in walking distance depends upon the degree of arteriosclerotic involvement in other muscles of the limb. The new pain that now limits progression is from a different site and, because of its distribution, it is often obvious that it arises from a different muscle. Infiltration of the skin or subcutaneous tissues overlying the tender patch does not alter the exercise-tolerance.

In order to deduce the muscle of origin from the history of surface-representation of pain, further experimental work was undertaken. A reliable method of producing experimental

muscle-pain is that described by Lewis and Kellgren (1939)—namely, the intramuscular injection of 6 per cent. hypertonic saline after infiltration of the skin and deep fascia with local anaesthetic. A few cubic centimetres is sufficient to cause deep aching, varying in intensity from discomfort to nauseating agony. The intensity depends not only on the susceptibility of individuals and the amount of solution injected, but also on the concentration of pain nerve-endings in the vicinity of the injection. These are probably sparse, so that the number of nerve endings that are stimulated varies by chance according to the site of the injected saline. It was our impression nevertheless that in patients with claudication, injection of tender areas, and mechanical stimulation by the needle-point, gave rise more readily to pain than similar procedures in " non-tender " areas and in normal muscles. Each muscle referred to in Figures 1 and 2 was injected in at least six different subjects, usually in both lower limbs. The areas of skin reference for the pain so produced were charted, and composite areas for many of the more important lower limb muscles were determined. The pain comes on almost immediately after the injection; it rises rapidly to maximal intensity; and it then falls gradually in four to six minutes. The quality is described characteristically as a " sickening ache " and is often compared to " cramp." There are ill-defined limits—a small centre of intense pain tending to shade off to mild aching at the periphery. Although limited in spread to the reference areas shown on the charts the pain may be felt far distant from the point of injection, and the site of maximal intensity may move within the area from minute to minute. When hypertonic saline is injected into muscles in the neighbourhood of joints (for example into the flexor hallucis longus or the extensor digitorum brevis near the ankle) the pain may be referred to the joint itself. When a peripheral mixed nerve such as the anterior or posterior tibial is stimulated by adjacent saline, paraesthesia is produced which is distinguished easily from the dull quality of " deep " muscle pain. The whole question of muscle pain is discussed admirably by Kellgren (1938).

CLASSIFICATION OF OBLITERATIVE VASCULAR DISEASE

It seems clear that the immediate cause of intermittent claudication is diminution of arterial supply to the muscles concerned; but the reason why the arterial supply is diminished is less apparent. There is much confusion in the terminology used in relation to obliterative arterial conditions causing circulatory deficiency in the lower limbs. Occlusive arterial disease beginning under the age of fifty years is usually classified as " Buerger's disease " or " thromboangiitis obliterans "; whereas over the age of fifty years the symptoms of arterial deficiency are usually grouped under the general term, arteriosclerosis. Leriche (1947), in a recent analysis of over 500 personal cases, limited the term thromboangiitis obliterans to patients under the age of thirty-five years; in patients over the age of fifty years he attributed the symptoms to arteriosclerosis; but he admitted that there was a third group of patients, with ages between thirty-five and fifty years, showing features of both conditions, that he was unable to classify.

Review of the arteriographic findings and clinical features in a large number of patients with deficiency of circulation in the lower limbs who were investigated between 1932 and 1949 show that they may be classified under three main headings: 1) primary thrombosis of the popliteal artery; 2) juvenile obliterative arteritis; and 3) senile obliterative arteritis—*a*) diffuse obliterative arteritis, *b*) secondary popliteal thrombosis, *c*) secondary femoral thrombosis (Figs. 3–14).

Primary thrombosis of the popliteal artery and juvenile obliterative arteritis are usually included together under the heading " thromboangiitis obliterans." It seems, however, that in most patients under the age of thirty-five years the cause of claudication is traumatic thrombosis and not arterial disease, and this was certainly true in every case in the series now reported.

Juvenile obliterative arteritis comprises a group of cases corresponding to those described by Buerger and this condition may be a pathological entity. Buerger himself included under the heading of thromboangiitis obliterans patients who, from their symptoms, appeared to be suffering from degenerative arterial changes. In this report the term " juvenile obliterative arteritis " is restricted to obliterative arteritis beginning in the feet and following a characteristic clinical course. The condition seldom, if ever, begins after the age of thirty-five years.

Senile obliterative arteritis is the term used to include the degenerative arterial changes that are commonly associated with increasing age and are included under the general term " arteriosclerosis." Arteriosclerosis is usually considered to be a disease of old age, but careful examination of post-mortem and biopsy material in obliterative arteritis from patients under the age of thirty-five years sometimes reveals arteriosclerosis at an early age. Well-marked calcification of the vessels has been seen in the early twenties. Arteriosclerosis is undoubtedly the most frequent cause of arterial deficiency in the lower extremities in the fourth decade, and over the age of forty years it accounts for most cases of occlusive vascular disease.

In brief, almost all cases of obliterative arteritis in young people up to the age of about thirty-five years fall into one of the first two groups—primary thrombosis of the popliteal artery and juvenile obliterative arteritis; and in patients over the age of thirty-five years into the third group—senile obliterative arteritis. The nature of the pathological changes in the blocked vessels is largely speculative, but it is of no great importance from the point of view of treatment which cannot be directed towards cure of the underlying condition or restoration of the function of blocked vessels. Attention must be concentrated on the vessels that are still healthy with the object of assisting by every possible means the greatest and most rapid development of a collateral circulation.

TABLE I

ANALYSIS OF 472 CASES OF OBLITERATIVE ARTERITIS OF THE LOWER LIMBS

	Men	Women	Total
Traumatic thrombosis . . .	4	1	5
Juvenile obliterative arteritis . .	8	—	8
Primary popliteal thrombosis . .	6	—	6
Senile obliterative arteritis . .	405	48	453
(*a*) Diffuse obliterative arteritis .	192	26	218
(*b*) Secondary popliteal thrombosis	165	15	180
(*c*) Secondary femoral thrombosis	48	7	55

PRIMARY THROMBOSIS OF THE POPLITEAL ARTERY

Nearly all patients under the age of thirty-five years who complain of intermittent claudication in the calf muscles have been found to have thrombosis of the popliteal artery. Arteriographic studies show that occlusion is confined to this artery, the other vessels appearing healthy. The extent of the occluded segment varies: it may extend from the level of the knee joint to the adductor opening; from the level of the knee joint to the termination of the artery; or throughout the whole length of the artery from the adductor opening to the bifurcation (Figs. 3–6). The fact that one end of the thrombosed segment is often found at the level of the knee joint suggests strongly that the causative process begins at this level. Several arteriograms obtained before there was actual thrombosis showed the earliest stage

FIG. 3

Primary popliteal thrombosis—pre-thrombotic lesion at the level of the upper border of the femoral condyles (from the Surgical Professorial Unit at St Bartholomew's Hospital by courtesy of Professor Sir James Paterson Ross).

of the lesion, namely, localised narrowing and irregularity of the lumen of the artery in the middle of the popliteal fossa (Fig. 3). Arteriograms of patients known to have sustained traumatic thrombosis of the popliteal artery from posterior dislocation of the knee joint (Fig. 7) are indistinguishable from primary thrombosis of the artery.

Histological examination of the occluded segment obtained by arterectomy from young men suffering from intermittent claudication of the calf muscles, usually thought to be due to " Buerger's disease," shows no evidence of inflammatory changes in the arterial wall (Boyd 1938). The lumen is found to be occluded by healthy clot in various stages of

FIG. 4

Primary popliteal thrombosis—thrombosis beginning at the level of the upper border of the femoral condyles has extended proximally as far as the tendon of adductor magnus (by courtesy of Professor Sir James Paterson Ross).

organisation. The wall of the artery is normal. Patients with this condition have been traced for ten years and there has been no evidence of further arterial disease. This is in striking contrast with the after-histories of patients with proved arterial disease ("thromboangiitis obliterans" or arteriosclerosis).

The evidence in favour of primary popliteal thrombosis as a clinical entity may thus be summarised: 1) there is similarity of the arteriographic picture with that of traumatic thrombosis from dislocation of the knee joint; 2) there is no inflammatory or degenerative change in the walls of the artery; 3) the clinical course is more suggestive of injury than

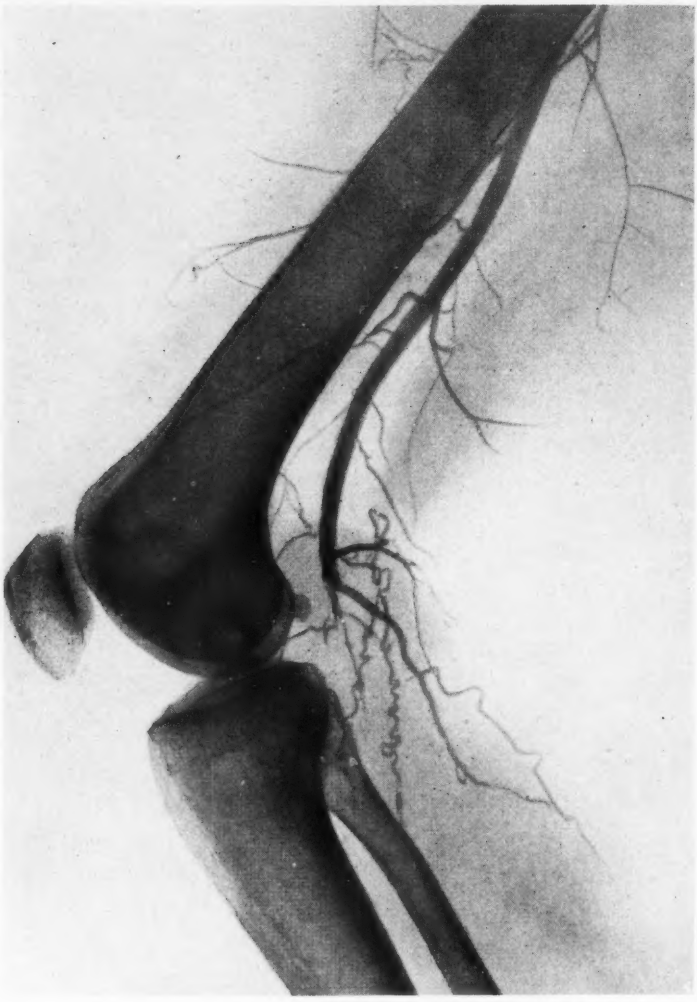


FIG. 5

Primary popliteal thrombosis—the occlusion has extended distally from the level of the upper border of the femoral condyles; proximal extension is probably limited by a large sural artery.

constitutional disease. In further investigation of this possibility, special studies of the anatomy of the popliteal artery were made (Boyd and Wilde 1949).

Anatomy of the popliteal artery—After leaving the adductor hiatus the popliteal artery lies in loose fatty tissue and is freely mobile. Just above the level of the knee joint the artery enters a fibrous tunnel derived from the fascia on the deep surface of the gastrocnemius. The fascial covering narrows to form a definite fibrous band, $\frac{1}{4}$ to $\frac{1}{2}$ inch broad, attached to the capsule of the knee joint at the level of the joint. Arteriograms show that it is at this point that thrombosis of the popliteal artery usually occurs. It is suggested that injury, from

FIG. 6

Primary popliteal thrombosis—thrombosis beginning at the level of the femoral condyles has extended both proximally and distally.

normal knee movement or minor sprains and strains, may cause damage to the tunica intima and thrombosis of the vessel. The possibility that there may be traumatic thrombosis is obviously increased if there is abnormal fixation of the popliteal artery to the oblique ligament within the fibromuscular canal from inflammatory changes caused by minor infection of the toes or interdigital folds.

Care should be taken to distinguish primary thrombosis of the popliteal artery from secondary thrombosis occurring as a complication of senile obliterative arteritis. This distinction is fundamental in assessing the prognosis. Senile obliterative arteritis often occurs

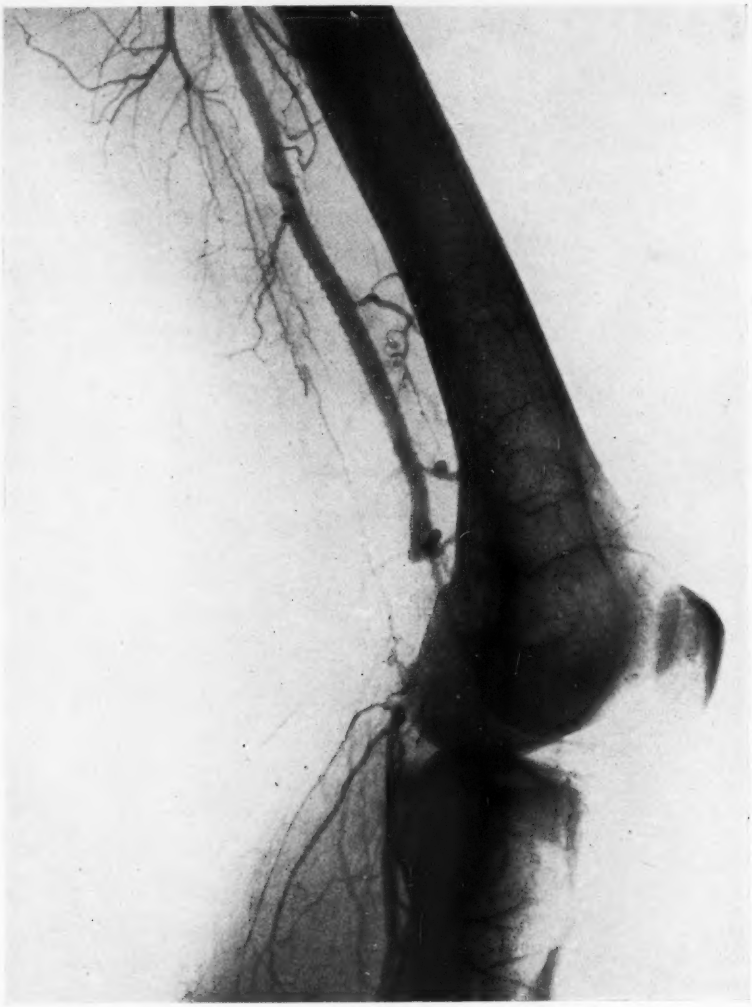


FIG. 7

Traumatic thrombosis of the popliteal artery from posterior dislocation of the knee joint. Note that the thrombosis begins at the level of the upper border of the femoral condyles and that the arteriographic appearances are identical with those of primary popliteal thrombosis in which there has been no major bone or joint injury.

in the fourth decade. On the other hand degenerative arterial changes may be delayed until much later in life. Occlusion of the popliteal artery in a man aged thirty-five years may not, therefore, be due to primary thrombosis; it may be secondary to early senile obliterative arteritis, the prognosis then being poor. A similar finding in a man aged fifty years, most commonly secondary to degenerative change, could possibly be primary if there was no generalised arteriosclerotic change, and in this event the outlook for both life and limb would be good. It is clearly important to determine by careful examination of the cardio-vascular system whether the thrombosis is primary or secondary.

JUVENILE OBLITERATIVE ARTERITIS

For the clinical syndrome in which obliterative arteritis begins distally in the arteries of the feet in young men, the general title " juvenile obliterative arteritis " is preferred to that of thromboangiitis obliterans, or Buerger's Disease. The first accurate study of " thromboangiitis obliterans," originally described by Winiwater in 1879, was published by Leo Buerger in 1908 and has since been known by his name. " Buerger's disease " like " Raynaud's disease " includes a number of conditions of differing pathology that have been separated from the general group of peripheral vascular disorders in which there is obliterative arteritis of distal distribution. The term " Raynaud's phenomena " is now used rather than " Raynaud's disease "; and similarly, for these conditions, some of which are due to arteriosclerosis, " Buerger's syndrome " would be more appropriate than " Buerger's disease."

After separating from this general group cases that are due to degenerative arteritis there remains an obliterative arteritis of obscure origin with characteristic clinical features that might be called " juvenile obliterative arteritis " or " thromboangiitis obliterans."

Juvenile obliterative arteritis seldom, if ever, begins after the age of thirty-five years. The obliterative process begins in the small arteries of the feet. Gradually, but relentlessly, it ascends the limb until the popliteal or even the femoral vessels are occluded (Figs. 8–10). There is often fungus infection of the interdigital folds. Patchy phlebitis in the superficial veins may precede the signs of arterial involvement—an occurrence that is seldom seen in arteriosclerosis. The pain usually becomes intolerable and by the time the obliterative process involves the popliteal artery amputation is demanded. The disease is often more advanced in one limb than the other but it is always bilateral. The more recently affected limb may need amputation even before the limb that was first affected.

In early cases, arteriography shows narrowing and finally obliteration of the small arteries of the feet. In more advanced cases arteriograms show relentless proximal spread of the disease; the tibial arteries, and eventually the popliteal arteries, become involved. A line can almost be drawn across the limb at the point to which the disease has reached. Collateral vessels show the corkscrew appearance that is associated characteristically with rapid hypertrophy and dilatation—a feature less marked in distal obliterative arteritis of arteriosclerotic origin.

Ischaemic pain and colour changes in the toes are often followed rapidly by ulceration and gangrene. Intermittent claudication in the small muscles of the sole of the foot, which unfortunately is often mistaken for the symptoms of chronic foot strain, may precede the more obvious phenomena of ischaemia. Ulceration occurs early, sometimes with gangrene, and not uncommonly in the presence of palpable pulses at the ankle. The course is progressive, though periods of apparent quiescence in one or other leg are often seen. One or both limbs are usually lost within three to five years of the onset of the disease.

It would appear that juvenile obliterative arteritis is confined to the lower limbs but this is an opinion which may need to be modified in the light of further experience. Involvement of the upper limbs is more typical of the distal type of senile obliterative arteritis.

Little help is gained from studying pathological changes in the vessels. Buerger, in his original study of nineteen amputated legs, described the macroscopic and microscopic changes which he considered to be specific. There was macroscopic evidence of perivascular fibrosis welding the artery, vein and nerve into a dense cord of fibrous tissue. Microscopy showed inflammatory infiltration of the vessel walls with fibrosis of the adventitia, atrophy of the media, and slight thickening of the intima from proliferation of endothelial cells. The lumen of the vessel was blocked by clots in various stages of organisation and, eventually, the thrombus became partly recanalised. These changes are seen also in arteriosclerosis. Any surgeon who has experience of amputating limbs for senile gangrene will have noticed

FIG. 8

Juvenile obliterative arteritis—early stage. Thrombosis in the plantar arteries extending as far as the necks of the metatarsals.

FIG. 9

Juvenile obliterative arteritis—thrombosis has extended proximally to the supramalleolar region.

occasional difficulty in separating the components of the neurovascular bundle which are often bound inextricably by fibrous tissue. The change is most marked if there has been sepsis with ulceration and gangrene. When there has been recent infection the tissue planes around the neurovascular bundle are oedematous. In late cases, the inflammatory tissue is organised and converted into dense fibrous cords. These changes give rise to difficulty when

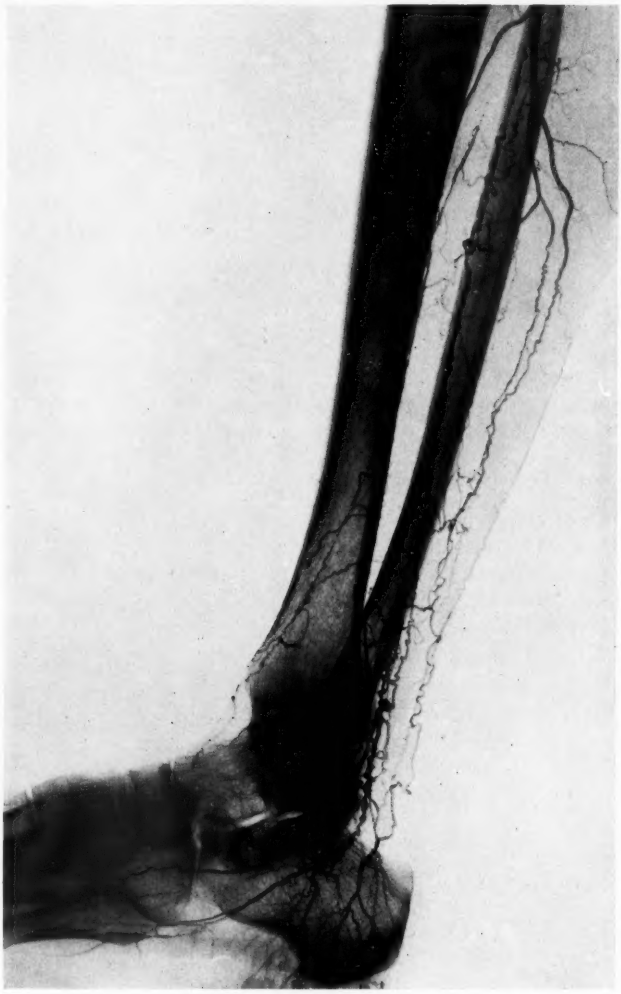


FIG. 10

Juvenile obliterative arteritis—late stage. Extension of thrombosis proximally. Thrombosis has nearly reached the bifurcation of the popliteal artery.

peripheral sympathectomy is attempted because isolation of the posterior tibial nerve from the artery and veins may present the greatest difficulty. The inflammatory change depends largely upon the degree of infection in the perivascular lymphatics. Specimens of the posterior tibial artery obtained by arterectomy in early cases, before there was infection and ulceration, do not show such marked perivascular fibrosis.

SENILE OBLITERATIVE ARTERITIS

This group includes the great majority of patients who complain of symptoms due to arterial deficiency. The degenerative arterial changes are most marked in the lower limbs. It is important to lay stress on the fact that senile obliterative arteritis is not confined to old age; it is seen in patients with wide variation of age. Marked atheroma has been recorded in young children, and it is by no means rare in the third decade; it is the most frequent cause of occlusive arterial disease between the ages of thirty and forty years; and it accounts for practically all cases of organic obliterative arterial change after the age of forty years.

a) Diffuse obliterative arteritis—Arteriography shows narrowing and irregularity of the main vessels with a beaded appearance of the larger branches (Fig. 11). Sometimes the pathological changes seem to fall principally on the main vessels; the smaller branches are abundant, and a collateral circulation is well developed. This is the most common group and there is often calcification of the arterial walls. In others, changes in the main vessels are less marked; the most noticeable features are the paucity of muscle branches which appear to end abruptly, the absence of collateral anastomoses, and the pronounced muscle wasting.

There is a third group, small but interesting, in which the most marked clinical feature is coldness of the feet and often of the hands. The toes, and sometimes the feet, are deeply cyanotic and cold but they become pink and warm when vasoconstriction is abolished by reflex heating or by paravertebral block of the lumbar sympathetic chain. There is little if any muscle wasting. Oscillometric readings are normal as far down as the ankle. In all three groups the blood pressure is usually high.

b) Secondary popliteal thrombosis—Thrombosis of the popliteal artery occurs in nearly half the patients with diffuse arterial disease. The arteriographic picture (Fig. 12) resembles that of primary popliteal thrombosis. Thrombosis begins in the segment of the popliteal artery behind the knee joint where the artery is attached to the capsule of the joint by a fibrous band. In diffuse obliterative arteritis arteriography shows the greatest changes in this segment. Repeated minor injuries caused by flexion and extension of the knee joint probably determine localisation of the disease to this part of the vessel. Thrombosis, beginning in the condylar region, usually extends upwards as far as the adductor opening (Fig. 13). Sometimes, however, distal extension occurs with occlusion of the lower half of the artery and the first few inches of the tibial vessels. Occasionally thrombosis extends in both directions, thus involving the entire length of the popliteal artery. The extent of thrombosis is probably determined by the calibre of branches leaving the artery, proximal spread being limited by the brisk flow through the anastomotica magna and distal spread by the flow through a large sural artery. The onset of thrombosis may be determined by injury or by lowering of the blood pressure with diminution of blood flow due to confinement to bed by reason of illness or operation. The effect of secondary popliteal thrombosis depends upon the degree to which the collateral circulation has already developed. If the calibre of the main vessels has been diminished gradually over a long period of time the collateral circulation is well developed and the effect of a new lesion on the peripheral circulation is negligible, but if thrombosis occurs early in the disease, severe ischaemia is bound to result.

c) Secondary femoral thrombosis—Thrombosis of the superficial femoral artery is much less frequent. In the series now reported it occurred in only fifty-five of 472 patients. Arteriography shows occlusion of the superficial femoral artery from the adductor opening to the point where the profunda femoris leaves the main trunk (Fig. 16). Thrombosis has been shown to begin in the region of the adductor opening, proximal extension being limited by the brisk flow of blood through the profunda femoris (Fig. 14). Arteriograms in diffuse obliterative arteritis often show gross deformity of the femoral vessel at the level of the adductor opening where the artery may be tethered to the margin of the tendinous opening.

FIG. 11

Senile obliterative arteritis—*a*) Diffuse obliterative arteritis.

The occurrence of femoral thrombosis is determined by much the same factors as those of popliteal thrombosis. The effect on the circulation in the limb depends, as in popliteal thrombosis, on the extent to which the collateral circulation has developed. Generally speaking the higher the block the less marked are the effects on the peripheral circulation.

Femoral thrombosis is to be suspected clinically if there is claudication in the calf of a limb which appears to be unusually healthy. Nutritional changes in the skin, nails and muscles—so commonly seen in secondary popliteal thrombosis—are seldom found when the femoral artery is occluded. Nevertheless, in femoral thrombosis, distal extension with gangrene of the limb occurs more commonly than in popliteal thrombosis.

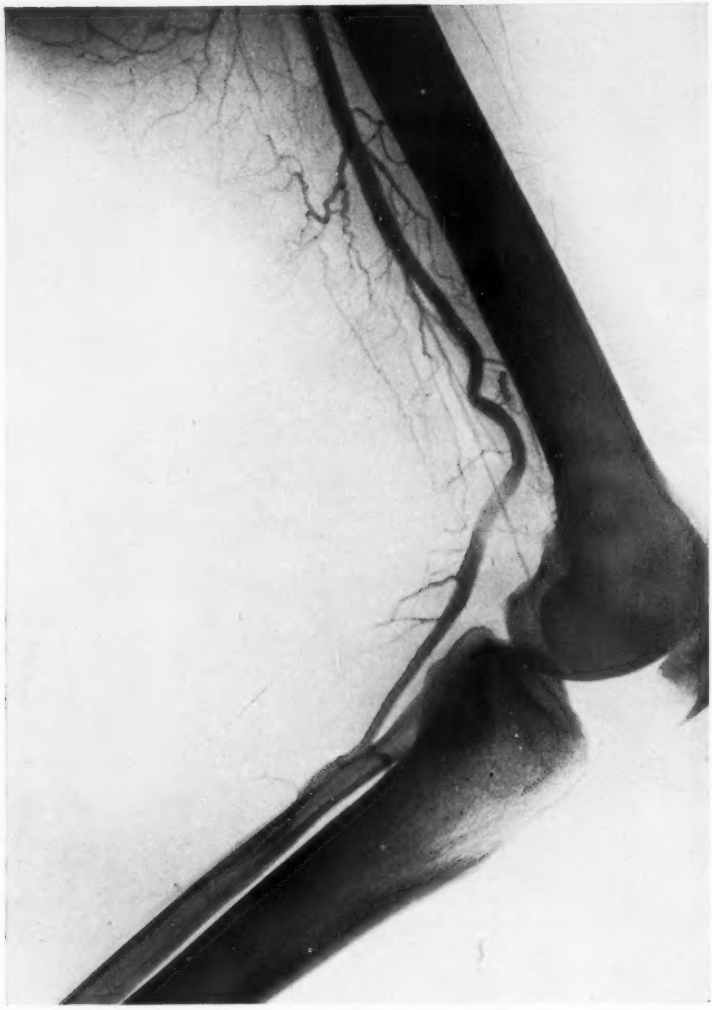


FIG. 12

Senile obliterative arteritis—*b*) Secondary politeal thrombosis. Early lesion beginning at the level of the upper border of the femoral condyles.

CLINICAL INVESTIGATION

Most patients in this series were first seen in the Neurovascular Out-patient Clinic of the Manchester Royal Infirmary. Investigation in the out-patient department was designed primarily to enable the surgeon to decide whether treatment could be given as an out-patient or whether it was advisable to admit the patient for further study. Peripheral vascular disease is of course part of a generalised degenerative change; the patient must be assessed as a whole, and it is not possible to carry out complete examination of the cardio-vascular system in a busy out-patient clinic; but before operative procedures were undertaken detailed

FIG. 13

Senile obliterative arteritis—*b*) Secondary popliteal thrombosis (note extension of thrombosis upwards as far as the opening of adductor magnus).

in-patient investigation was always made. Salient features in the history and clinical findings were recorded on a printed proforma which recorded the location of pain, the approximate date when it was first noticed, the suddenness of onset, the distance walked at the time of onset and at the time of examination, and the presence or absence of coldness, numbness or rest pain. A record was made of history of injury, frostbite or phlebitis, of the amount of tobacco smoked, and of the age and cause of death of the parents. Clinical data included records of the general health, the state of the heart and blood pressure, the nutrition of the limb as estimated on an arbitrary scale—Grades I, II and III—the presence or absence of

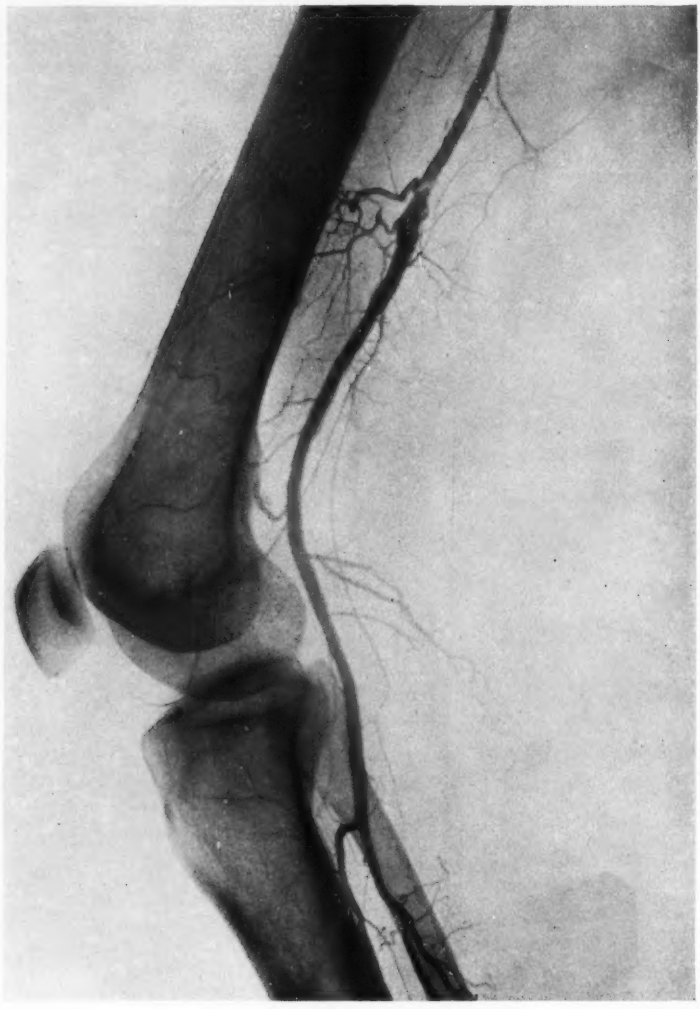


FIG. 14

Senile obliterative arteritis—*c*) Secondary femoral thrombosis—early lesion at the level of the adductor opening.

muscle wasting, atrophy of the small muscles, atrophy of the nails, and colour changes. Records of walking-ability included the rate of walking, the site of pain after walking, the time of onset, the time of halting, the radiation of pain, and the relation of walking to diminution or stabilisation of pain. The pulses and oscillometric readings in both lower limbs were recorded on a chart. Oscillometric readings were taken above and below the knee, and above the ankle. The oscillometer is not an instrument of precision but it does allow assessment of changes in the pulsation of main vessels. Reduced oscillations throughout the limb are characteristic of diffuse obliterative arteritis; complete absence of oscillation is

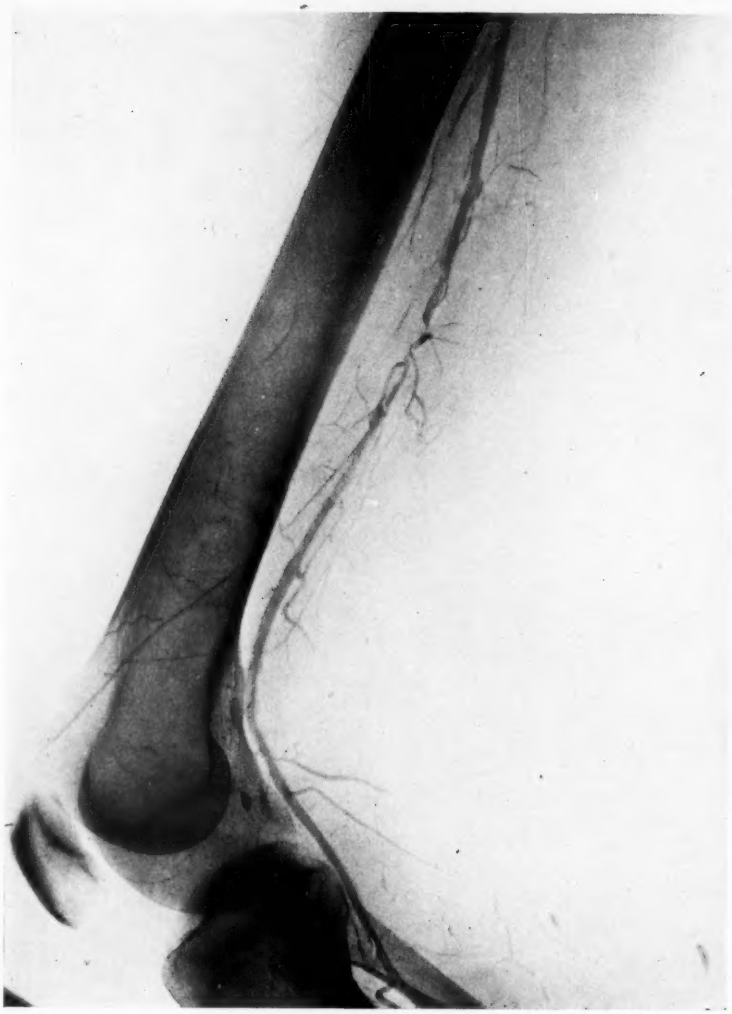


FIG. 15

Senile obliterative arteritis—illustrating the progress of a secondary femoral thrombosis.

usually found below a main vessel block, though it is to be noted that if the collateral circulation is well developed there may be slight excursion of the needle. One of the authors (A. M. B.) has used the oscillometer for fifteen years and during that time arteriography of doubtful cases has never revealed evidence of main vessel block that was not suspected by oscillometry.

Investigations thus outlined determine the underlying cause of claudication; and the clinical history of the localisation of pain indicates which muscles are involved. It remains to confirm the observations of the patient by examining his reactions to measured degrees

FIG. 16

Senile obliterative arteritis—*c*) Secondary femoral thrombosis—fully developed secondary thrombosis of the superficial femoral artery.

of exercise, and also to assess the severity of the condition. The circulatory deficiency must be estimated in terms of the demands made by the individual upon his blood supply. The primary concern of a patient is the interference with his daily life caused by pain in the leg and, when the limb is not in danger, treatment must be directed to the relief of subjective manifestations. Estimates of the relative deficiency were therefore made by testing the ability of each patient to walk at his own rate under controlled conditions. This was done first by walking over a known circuit of the hospital corridors; but the test has now been replaced by walking on a machine specially constructed for the purpose (Fig. 17). An endless

belt, driven by an electric motor at a speed ranging from one to four and a half miles per hour, is fitted with a tachometer giving the speed in miles per hour and the distance travelled by a point on the belt. The patient steps forward from a stationary platform on to the belt which is moving towards him at the slowest speed; and the speed of the belt is then increased gradually until he believes that he is walking at a normal rate.

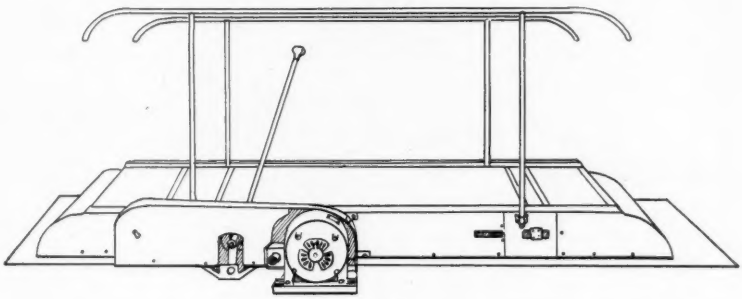


FIG. 17

The claudicometer—exercise tolerance machine.

Such a machine permits direct observation of the limb throughout the test. It has been noted that sometimes there is pallor of the feet at the onset of claudication. Oscillometry after the exercise-period sometimes shows reduced readings, contrary to the usual finding of a marked increase. During the exercise-test the patient describes his sensations. From the data thus obtained it is possible to classify the type of arterial disorder.

CLASSIFICATION INTO CLINICAL TYPES

The hypothesis that there are three clinical types of claudication depends upon consideration of the events taking place in a muscle during exercise. The resting muscle has a scanty blood supply; but when it is exercised there is capillary vasodilatation (Krogh 1922). If the muscle is exercised under ischaemic conditions there will be pain (Lewis, Pickering and Rothschild 1929). It cannot be doubted that the protatic event in these phenomena is the accumulation of metabolites—both in the case of vasodilatation and of pain (Hamilton 1947, Lewis 1942). It is beyond the scope of this paper to discuss the mechanism involved, the nature of the various metabolites concerned, or their mode of action. The basic concept is not affected.

With the onset of muscle-exercise, metabolites accumulate until the threshold for the reaction of vasodilatation is reached, such increase in blood supply dissipating the metabolites by chemical or physical processes. Eventually equilibrium is reached—the metabolites being eliminated at the same rate that they are produced. The level at which this takes place depends, for a given rate of exercise, upon the available blood supply. In the normal individual equilibrium is reached before the metabolites accumulate sufficiently to produce pain. In the patient with claudication the threshold of pain-reaction is reached before equilibrium is attained. Figure 18 is an attempt to express this diagrammatically; the horizontal axis represents time from the beginning of exercise; the vertical axis represents accumulation of metabolites; curve (a) is that of a normal individual; curve (b) is that of a patient with claudication. The dotted lines show the levels of metabolites required to initiate the increase of blood supply (" vasodilatation ") and the pain reaction.

Most patients with intermittent claudication find that when pain begins it is relieved quickly by a short period of rest and thereafter they can go on walking. In such cases,

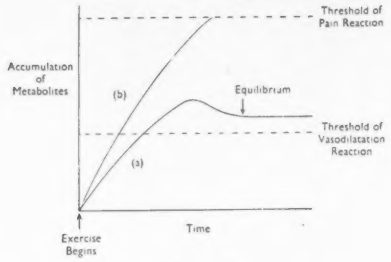


FIG. 18

Curves representing reaction to exercise in the normal individual (curve (a) and the patient with claudication (curve (b).

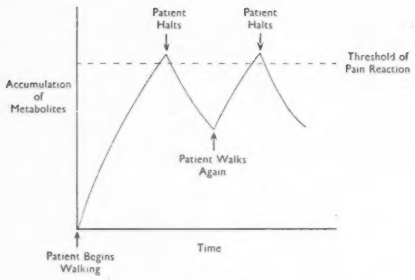


FIG. 19

Alternating exercise and rest in intermittent claudication.

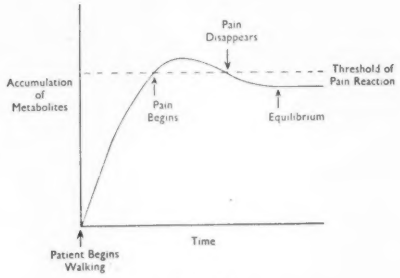


FIG. 20

Reaction to continued exercise in Type 1 claudication.

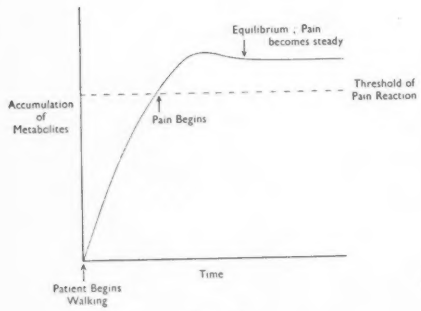


FIG. 21

Reaction to continued exercise in Type 2 claudication.

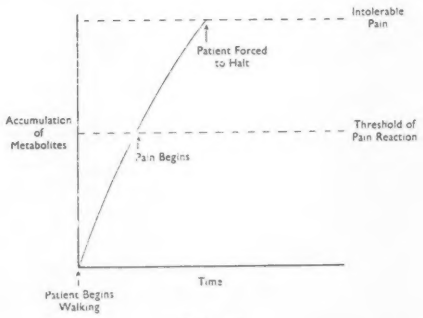


FIG. 22

Reaction to continued exercise in Type 3 claudication.

metabolites were accumulated until the threshold was reached and they were dissipated during the rest period, only to rise again when walking was resumed (Fig. 19).

When a patient is asked to walk for the purposes of investigation he is not allowed to rest when pain first develops; he is urged to continue walking as long as possible. The events that occur after the onset of claudication vary according to the relationship of blood supply to the demand and three types of reaction may be distinguished. *Type* 1—In this type the

blood supply and demand are very nearly equal. When the patient is urged to continue walking he announces, usually in a surprised tone, that the pain has disappeared. Here the level of metabolites crosses the pain threshold before a position of equilibrium is reached; but when equilibrium is finally attained it is below the threshold (Fig. 20). The fact that equilibrium has been attained just below the threshold of pain can be demonstrated by making the patient walk more quickly than usual; pain then returns. *Type* 2—Most patients belong to this group. When the patient continues walking the pain attains a steady level and eventually he stops walking not because of the intensity of the pain but because of its persistence. Equilibrium is attained only above the threshold for pain-reaction (Fig. 21). *Type* 3—Here the blood supply is so far reduced that stability cannot be achieved; on being urged to walk further the patient complains that the intensity of pain is increasing; finally it becomes intolerable so that he is forced to halt. In this case the position of equilibrium is at so high a level that it cannot be reached because in attempting to achieve it there is unbearable pain (Fig. 22).

TREATMENT OF INTERMITTENT CLAUDICATION

General considerations—If traumatic thrombosis is excluded, the arterial condition that underlies intermittent claudication is widespread, progressive and ultimately fatal. Lack of knowledge of the etiology of the responsible arterial conditions limits the treatment of intermittent claudication to alleviation of the subjective manifestations that interfere with the daily life of the patient. The most rational method of relieving exercise-pain would be to increase the blood supply in order that the demand of exercising muscles might be met. When the deficit is small this can be done; but in severe cases the gap between supply and demand may be too great, or it may not be possible to increase the blood supply because there is severe occlusive change in a group of muscular vessels. Under these conditions relief can be gained only by reducing the function of the affected muscles.

In practice the problem is more complex than one of simple supply and demand. Each individual case must be considered carefully and many factors of varying importance must be assessed accurately, always making allowance for the fact that many patients are poor witnesses and that it is not always easy to determine the degree to which an individual is inconvenienced by claudication, or indeed whether he is claudicating at all.

Factors influencing the choice of treatment—1) *Clinical type*—In determining treatment, the clinical type of claudication is by far the most important factor and this cannot be over-emphasized. Failure to appreciate that there are different grades of claudication has led, on the one hand, to extravagant claims for the success of certain drugs or methods of treatment and, on the other, to disrepute of sound and logical procedures. The general principles of treatment that are indicated in each clinical group can be defined clearly. *Clinical Type* 1—In this type, supply and demand are almost equal. The very slightest increase in blood flow brings about complete relief of exercise-pain and there is a good response to any method of treatment that improves the blood supply. Suggestion is a powerful factor in any form of treatment and it may have particular application to patients who suffer from claudication. If the patient is a Type I claudicant he will respond to almost any method of treatment, provided only that his confidence is gained, and this accounts for the reputation that has been gained for Buerger's exercises, intermittent venous occlusion, and the administration of muscular extracts, hormones and anti-coagulants. *Clinical Type* 2—In this type of claudication the gap between supply and demand is much wider and relief can be gained only by bridging the gap. As a rule the supply is increased to meet the demand, but in certain cases the demand may be reduced to meet the supply. Sometimes a combination of both methods may be adopted. *Clinical Type* 3—In this type the gap between supply and demand can be narrowed but never closed—a fact that is of fundamental importance. Disappointment is

bound to result from operative treatment or from any measure that is designed solely to improve the blood flow. Relief from severe pain can be secured only by reducing the function of the muscles by neurectomy or tenotomy.

2) *Age*—The age of the patient has an important bearing on treatment; the younger he is the more necessary is it to secure the greatest possible increase in blood supply. Generally speaking, lumbar sympathectomy should be advocated in patients under the age of forty to fifty years regardless of the clinical category in which they may fall, not only because in younger patients it may be possible to improve the general nutrition of the limb, and by attention to general health to delay the progress of arteriosclerotic change, but also because the longer the patient has to live the more likely it is that amputation will be needed sooner or later.

3) *Type of arterial disease*—Estimation of the severity and type of the causative arterial condition is of great importance in planning treatment. In traumatic thrombosis the expectation of life is normal and the only danger is that of local failure of the collateral channels to undergo sufficient hypertrophy and hyperplasia. The most vigorous and dependable method of increasing blood supply and encouraging the collateral circulation is indicated. The questions that arise are: what is the risk to life, and what is the risk of losing the limb? *The risk to life*—Experience during the last eighteen years has shown that in diffuse obliterative arteritis, with calcification of the main vessels, loss of life is more likely than loss of the limb. These patients often suffer from hypertension, coronary thrombosis, left heart failure and cerebral complications; sometimes there are mesenteric vascular accidents. Calcified vessels associated with hypertension show little tendency to thrombosis, and peripheral gangrene is unusual. In general, angina of effort, past history of coronary thrombosis, dyspnoea on exertion, and hypertension are contra-indications to lumbar sympathectomy. Lumbar sympathectomy should be advised only in patients with diffuse obliterative arteritis and calcification of the main vessels when it is felt that the limb itself is in danger. *The risk of losing the limb*—Peripheral gangrene leading to loss of the limb is more likely to occur in obliterative arteritis complicated by secondary thrombosis of a main vessel, especially when the superficial femoral artery is involved. Patients with secondary thrombosis of the superficial femoral artery nearly always have a normal or low blood pressure and are specially prone to massive thrombosis followed by extensive gangrene. In Type 3 claudicants, where no more than a pain-relieving procedure is indicated, an effort to improve the blood flow should always be made. This applies also to patients with superficial femoral thrombosis.

4) *Other painful conditions in the limb*—It is not surprising that claudication in patients who are past middle-age, brought about by degenerative arterial changes, is often associated with other signs of wear and tear of the tissues. The clinical picture is often complicated by osteoarthritis, chronic foot strain, deep thrombo-phlebitis or varicosity of the veins. The contribution of each of these conditions to the disability must be estimated. It is clearly of no value to gain operative relief from claudication if, in fact, the pain was due to osteoarthritis of the knee joint. Much help can be gained in differentiating joint, muscle and vascular pains by infiltration of the popliteal or tibial nerves with novocain, and by paravertebral block of the lumbar ganglia. By repeating walking tests after the various nerve blocks it is usually possible to determine the relative importance of each source of pain.

5) *Influence of associated disease*—Chronic bronchitis and emphysema, angina of effort, and obesity, are often associated with arteriosclerosis and intermittent claudication; and operations for the relief of intermittent claudication should not be undertaken if, in fact, the halting of the patient is due to dyspnoea. The importance of these conditions can best be determined by direct observation under walking conditions. Diabetes, unless mild and well controlled, worsens the prognosis and accounts for rapid progress in the arteriosclerotic changes and the development of peripheral gangrene. Diabetes must be excluded before decisions are made as to treatment.

6) *Economic and geographical factors*—The necessity for immediate and complete relief of pain may arise from the inability of a patient to walk long distances to and from his work; and sometimes the contours of the country in which he lives may be a determining factor. A wealthy patient, with car and chauffeur, who is brought to a halt at 150 yards may have no need, and indeed no inclination, to walk more than 100 yards and he can be treated palliatively; whereas a patient able to walk 400 yards who is compelled to walk half a mile to his work requires direct attack on the affected muscle.

Details of treatment—Buerger's exercises, contrast baths, intermittent venous occlusion, suction-pressure and similar measures of treatment had been prescribed in many cases included in this series before we first saw them but without any real evidence of improvement. We believe that they are of little value and have not used them. The methods of treatment that have been employed will be discussed under the headings: 1) methods of improving the blood supply; 2) methods of decreasing the vascular demands of muscles; 3) methods of relieving pain. The results, with brief comments, are tabulated at the end of each paragraph.

The classification of results calls for a word of explanation. Success claimed for 100 per cent. improvement might in practice mean no more than an increase in walking distance from one to two hundred yards. It was decided therefore to classify results under three headings: *Good*, meaning complete relief from pain or ability to walk one mile without pain; *Improved*, indicating that the patient was able to walk a useful distance, arbitrarily fixed at half a mile, without pain; *Unrelieved*, including patients in whom the walking distance was often much increased but was still not accepted as being of economic value.

METHODS OF IMPROVING THE BLOOD SUPPLY

Lumbar ganglionectomy—The quickest, the most radical, and in general the most satisfactory way of improving the blood supply is by sympathectomy. Evidence of calcification of the main vessels is no contra-indication to sympathetic denervation. Some of the most successful results of sympathectomy in senile obliterative arteritis have been gained in patients with radiographic evidence of calcification of the main vessels. Release of normal tone increases the blood flow even if there is no evidence of abnormal vasoconstriction. Generally speaking, lumbar ganglionectomy should be undertaken in all patients below the age of sixty years if the general condition does not contra-indicate operation. The first, second and third lumbar ganglia, with the intervening chain, should be removed in order to be certain that denervation is complete. The more usual operation, in which only the second and third ganglia are removed, is apt to result in incomplete denervation (Jepson and Ratcliffe 1949).

In patients over the age of sixty years lumbar ganglionectomy is not always advisable but if the general cardio-vascular condition is satisfactory there seems no reason why sympathectomy should not be undertaken even in these older patients. If it is believed that the risk of operation is not justified, chemical destruction of the sympathetic chain with 10 per cent. phenol is worth considering (Haxton 1947).

TABLE II
RESULTS OF GANGLIONECTOMY IN SIXTY-THREE CASES

	Good	Improved	Unrelieved
Type 1 . . .	1	—	—
Type 2 . . .	36	11	5
Type 3 . . .	—	4	6

Comments on treatment by lumbar ganglionectomy—The results of lumbar ganglionectomy for arteriosclerosis confirm our previous views that this simple operation offers the best chance of rapid and certain improvement. The patient is allowed to get up on the fourth day and is discharged from hospital on the eighth or ninth day after operation. In many cases, walking

tests carried out on the day that they are discharged from hospital show great improvement. In others there is little immediate change but review one month later shows improvement. The distance that they can walk without pain steadily increases until most of them can walk a mile or more in comfort. The operative mortality is low. In this series, there was one death from coronary thrombosis on the ninth post-operative day. Altogether 632 lumbar ganglionectomies have been performed between January 1947 and March 1949 with one death. Two patients had cerebral thrombosis within forty-eight hours of operation but both recovered completely within a few weeks.

Paravertebral block with 10 per cent. phenol—Chemical destruction of the lumbar ganglia has proved to be of great value in patients in whom lumbar ganglionectomy was contra-indicated by reason of advanced age or impairment of general condition. The use of alcohol has been abandoned almost entirely on account of the not infrequent occurrence of muscular palsy and severe neuralgia due to unavoidable inclusion of the spinal nerves in the field of injection. Mandl (1948) showed in cats that 6 per cent. phenol had a selective action on the sympathetic ganglia causing complete destruction without damaging the spinal nerves. H. A. Haxton (1947), working in the Neurovascular Unit at the Manchester Royal Infirmary, suggested 6 per cent. phenol for paravertebral block of the lumbar chain. Haxton's early cases, in which he used 5 cubic centimetres of 6 per cent. phenol, showed evidence of incomplete sympathetic denervation without undesirable sequelae. In order to accomplish more complete destruction of the lumbar ganglia he increased the strength of the phenol solution to 10 per cent., and the quantity injected to 10 or 15 cubic centimetres. Paravertebral block with 10 to 15 cubic centimetres of 10 per cent. aqueous solution of phenol has been used in this Unit in a large number of patients since May 1947, with gratifying results. So far no complications of any sort have been met.

Technique of paravertebral block—The equipment needed is a 10 c.c. Labat syringe, a fine needle for intradermal injection, 12 cm. and 16 cm. rustless steel needles 0·8–1 mm. diameter, 2 per cent. novocain, and 10 per cent. aqueous solution of phenol. The injection can be carried out easily in bed. The patient lies on his side with a pillow under the loin in order to separate the lumbar transverse processes widely. It is important that the back should be kept straight, avoiding either flexion or extension, and that the trunk should be in a strict lateral position at right-angles to the bed. The foot of the bed is raised on 10-inch blocks in order to encourage seepage of the phenol upwards along the tissue planes around the sympathetic chain, thus reaching the first lumbar or even the twelfth dorsal ganglia.

The bedclothes should be removed so that the lower extremities are exposed to room temperature, about 20° C. being ideal. The skin temperature of the feet, preferably on the inner side of the heel, is recorded, readings being taken every few minutes until the skin temperature reaches a steady level. After skin preparation and isolation of the lumbar region with sterile towels, an intradermal wheal is raised with 2 per cent. novocain at a point near the outer border of the erector spinae, four fingers breadth lateral to the spine of the second lumbar vertebra. A 16 centimetre needle is most commonly used but a 12 centimetre needle is adequate in small and thin individuals. The needle is passed obliquely through the selected point, directed medially at an angle of about 30 degrees from the horizontal plane. The operator should make a mental picture of the relations of the erector spinae, psoas muscle and vertebral bodies, drawing an imaginary line from the point of injection to the front of the body of the second lumbar vertebra.

By this technique the needle often passes lateral to the tip of the transverse process. If possible the transverse process should be felt, the needle be withdrawn a little, inclined slightly upwards or downwards in order to pass above or below the bone, and then advanced a further 4 or 5 centimetres through the psoas muscle until the antero-lateral aspect of the vertebral body is reached. Occasionally the needle passes in front of the vertebral body and pierces the aorta or vena cava. Puncture of the great vessels is harmless and is, in fact, a useful indication of the position of the needle point. If this occurs the needle should be withdrawn and reinserted at a greater angle until bone is felt. When the needle is judged to be placed satisfactorily in close proximity to the lumbar chain, after careful aspiration in order to be quite certain that the spinal theca or a blood vessel has not been entered, 2 cubic centimetres of buffered 2 per cent. novocain solution is injected.

If the needle has been placed correctly a rise of skin temperature, usually first detectable in the skin over the medial side of the heel below the medial malleolus, will be recorded within two or three minutes. A large rise in the skin temperature cannot be expected in patients with advanced arteriosclerosis. In

any patient, however, in whom phenol block is indicated, there will be some elevation of skin temperature. A rise of even one degree, occurring within two or three minutes, shows that the needle is correctly placed.

If there is no alteration in skin temperature within five minutes, the needle should be withdrawn and reinserted at a different angle and the novocain injection repeated. It is unwise to inject the phenol solution unless the point of the needle is proved to be correctly placed. Only when the operator is satisfied with the position of the needle should 10 to 15 cubic centimetres of 10 per cent. aqueous solution of phenol be injected. The phenol may not be in complete solution at room temperature. If the fluid is cloudy, the bottle should be warmed by standing it in hot water for a few minutes until the phenol is completely dissolved. The syringe also should be warm.

The patient should remain in the lateral position for twenty minutes in order to keep the pool of phenol in contact with the lumbar ganglia. He may then be turned on his back and the blocks removed from the foot of the bed. He should remain flat on his back for one hour, after which he can return home with instructions to lie down for the rest of the day.

TABLE III
RESULTS OF PHENOL INJECTION IN TWENTY-SEVEN CASES

	Good	Improved	Unrelieved
Type 1 . . .	3	—	—
Type 2 . . .	5	9	4
Type 3 . . .	—	—	6

Comments on treatment by paravertebral block with phenol—Chemical sympathectomy with 10 per cent. phenol has a definite though limited place. The method has been used in patients over the age of sixty years and in those in whom lumbar ganglionectomy was contra-indicated by reason of their general condition. Paravertebral block with phenol has been used also in advanced Type 3 cases where the patient complained of a cold foot or where the feet showed severe nutritional changes or incipient gangrene.

Vitamin E (α-tocopherol) therapy—It is unfortunate that the use of α-tocopherol in cardio-vascular conditions has been exploited by the popular press as a new "miracle drug." Critics have applied the principle "*Falsus in uno; falsus in omne*" and thereby discredited favourable observations. The treatment was supported by Vogelsang and Shute (1947, 1948) and criticised by Baer and Heine (1948, 1949). The authors did not feel convinced by the arguments presented on either side and decided that observations should be made on selected cases. Vitamin E was given to Type 2 cases who were awaiting admission to hospital. Ten patients were given daily doses of 200 milligrammes of α-tocopherol (Ephynal, Roche). Their progress was reviewed after one month. They stated that they felt better but assessment of walking-ability showed little if any change. In every case, however, there was noticeable objective improvement after two months. The dose of "Ephynal" was increased to 400 milligrammes daily with further improvement. Larger doses have been used since, but it would appear that a daily dosage of 400 milligrammes is optimal. In view of these results it was considered that further observations were warranted. Vitamin E has also been given with benefit to patients with severe nutritional changes in the feet.

TABLE IV
RESULTS OF TREATMENT WITH α-TOCOPHEROL IN EIGHTY-ONE CASES

	Good	Improved	Unrelieved
Type 1 . . .	5	—	—
Type 2 . . .	27	32	13
Type 3 . . .	—	—	4

Analysis of the cases treated with α-tocopherol is shown in Table IV. It must be emphasized that these figures are derived only from clinical observation. The number of variables is such that strictly controlled experiments must of necessity involve long term studies; these are being undertaken and the results will be reported in due course.

Comments on treatment by α-tocopherol therapy—α-tocopherol is the only substance that has given consistently good results in cases other than those grouped in Type 1. Patients with vascular disorders of Type 2 and 3 who had previously been treated with other drugs without benefit showed definite improvement when treated with "Ephynal." The consistency with which there was a lag period of four to six weeks before improvement was noted was most striking. After a few months there was much improvement in the appearance of the feet in patients who showed nutritional changes.

METHODS OF DECREASING THE VASCULAR DEMAND BY MUSCLES

Thiouracil—A trial of thiouracil in the treatment of intermittent claudication was initiated by a chance observation. A patient attended the Vascular Clinic of the Manchester Royal Infirmary who had suffered for many months with Type 3 claudication and was also severely thyrotoxic (B.M.R.+50 per cent.). It was decided that the thyrotoxicosis should be controlled with thiouracil before treatment of the claudication was undertaken. When a normal B.M.R. had been reached, and the clinical manifestations of hyperthyroidism were controlled, it was discovered that the pain of claudication was greatly relieved.

TABLE V
RESULTS OF TREATMENT BY THIOURACIL IN THIRTY-ONE CASES

	Good	Improved	Unrelieved
Type 1 . . .	1	—	—
Type 2 . . .	6	1	16
Type 3 . . .	—	—	7

Thirty-one patients with intermittent claudication of mixed types, whose B.M.R.'s and blood cholesterols were within normal limits, were then treated by a prolonged course of thiouracil until a state of early myxoedema, confirmed by B.M.R. and blood cholesterol readings, was reached.

Comments on treatment by thiouracil—No significant improvement was noted in this group as a whole, even in cases that developed severe myxoedema. Our conclusion is that thiouracil is not warranted as a form of treatment for intermittent claudication.

METHODS OF RELIEVING PAIN

Antistin—The work of Barsoum and Gaddum (1935) suggested that in the ischaemic limb the histamine content of venous blood was increased. This statement has not been accepted generally but it seemed wise to try the effects of anti-histamine drugs to see if relief could be afforded to patients with claudication. Antistin was selected for trial because it appeared to

TABLE VI
RESULTS OF ANTISTIN THERAPY IN TWENTY-SIX CASES

	Good	Improved	Unrelieved
Type 1 . . .	5	—	1
Type 2 . . .	—	4	13
Type 3 . . .	—	—	3

have the least undesirable side-effects; and it was given to twenty-six patients in a daily dosage ranging from 300 milligrammes to 900 milligrammes according to tolerance. There were many unpleasant side-effects including headache, nausea and vomiting. One patient lost his sense of smell but recovered it when administration of the drug was stopped. There were two cases of cerebral thrombosis but these may have been coincidental. In view of the variable tolerance and the high incidence of side-effects, the treatment was discontinued.

Comments on antistin therapy—The mixed properties of the anti-histamine compounds at present available make it very difficult to assess their mode of action in intermittent claudication. The unpleasant side-effects make their use undesirable.

Myoneurectomy—*Internal popliteal neurectomy*—The possibility of relieving the pain of intermittent claudication by division of branches of the internal popliteal nerve to the gastrocnemius occurred to Sir James Learmonth and ourselves independently and at about the same time. While one of us (A. M. Boyd) was in charge of a Centre for Vascular Injuries in Egypt during the recent war it was observed that whereas ligation of the popliteal vessels usually gave rise to intermittent claudication, some patients were still able to walk without pain after excision of an aneurism in the popliteal fossa. It was noted that patients who did not complain of the pain of claudication had paralysis of the gastrocnemii, doubtless through division or stretching of branches of the internal popliteal nerve during exposure of the aneurism. The significance of this observation was not appreciated until 1947 when an arteriovenous aneurism of the popliteal artery was operated upon and the gastrocnemius was paralysed. Thereafter it was decided to divide the nerves to both heads of the gastrocnemius in patients with severe claudication.

In practice, the operation proved disappointing, although much interesting information was obtained. Patients who were relieved of the pain of claudication in the gastrocnemius nevertheless suffered pain in the soleus after walking a further hundred yards or so. The necessity of knowing the skin reference of pain from various muscles of the limb was thus revealed and for this reason studies of the cutaneous reference of pain from these muscles were undertaken (*vide supra*).

Division of the external popliteal nerve—Three patients complained of severe exercise pain in the extensors of the leg which halted them after walking 100 yards or less. The external popliteal nerve was infiltrated with 2 per cent. novocain and walking tests were then repeated. All three were relieved of pain and were able to walk more than half a mile in comfort. They did not mind the foot-drop and all agreed that the external popliteal nerve should be divided. The nerve was exposed through a half-inch incision over the neck of the fibula; it was crushed and divided. All three patients were able to walk painlessly on the second day after operation. So far they have remained satisfied.

Division of the posterior tibial nerve—A lady aged sixty years, weighing 18 stone (252 pounds) complained of severe pain in the left calf and in the sole of the left foot under the heads of all the metatarsals; it was difficult to determine which pain was the more crippling. There was diffuse obliterative arteritis and early nutritional changes in the foot. Paravertebral block with 10 per cent. phenol relieved the exercise-pain in the left calf, but the patient was halted at seventy yards by pain under the heads of the metatarsals. The posterior tibial nerve at the level of the ankle joint was infiltrated with 2 per cent. novocain, after which she was able to walk a quarter of a mile in comfort, being halted only by dyspnoea. Division of the nerve just above the ankle joint gave complete relief.

Tenotomy of the tendo Achillis—Sir Heneage Ogilvie (1949), in his Beyer Memorial Lecture, drew attention to the value of occasional idleness. In this erudite address, Ogilvie stressed the necessity of having time to think. One of us (A. M. B.) enjoyed such a period of idleness in an inn in Somerset, listening to local gossip, and in that time the possible value of tenotomy of the tendo Achillis occurred to him. He learned that in the latter part of the nineteenth century a general practitioner in that vicinity did all his rounds on horse-back and kept his horses until they were too lame to work, whereupon he divided a tendon near the hoof and thereby gained from them another five years of work. Horses are prone to claudication and it seemed that relief was obtained by dividing the tendon of the affected muscles.

After consultation with our orthopaedic colleagues we decided that division of the tendo Achillis might relieve exercise-pain in the calf muscles. The first patient upon whom this operation was performed was one with Type 3 claudication with exercise-pain in the right

calf which halted him at seventy yards. After division of the tendo Achillis he was able to walk two miles without pain on the third day after operation. He is now walking five miles daily in a hilly district in Wales carrying on his job as a postman. The operation of subcutaneous tenotomy of the tendo Achillis can be carried out in a few seconds under pentothal anaesthesia. The tendon is divided with a fine sharp tenotome just above the heel. The tenotomy knife is inserted on the medial side and the tendon is divided from within outwards. There is no bleeding. The patient is able to get up the next day and most of them are able to walk half to one mile; others complain of pain at the site of operation and defer walking until the second or third day. By the fourth day after operation all patients are able to walk more than one mile without pain. There have been no complications and no serious disabilities after operation. The gait is almost normal and not noticeably worse than before operation. Plantar-flexion is of course weakened but most patients are able to rise on their toes. After a few months the severed ends of the tendon become united with lengthening, but the patients are still free from exercise-pain.

TABLE VII
RESULTS OF TENOTOMY OF THE TENDO ACHILLIS IN TWENTY-FOUR PATIENTS

	Good	Improved	Unrelieved
Type 1 . . .	—	—	—
Type 2 . . .	6	—	—
Type 3 . . .	16	2	—

Comments on treatment by tenotomy of the tendo Achillis—This simple but eminently satisfactory operation has replaced popliteal myoneurectomy in this clinic. The operation was originally restricted to Type 3 cases. It was felt, however, that the results were so satisfactory that late Type 2 patients, whose improvement by other methods had been inadequate, might be included.

GENERAL COMMENTS

The most successful treatment for the Type 3 case of intermittent claudication, in which the blood supply is so far reduced that stability cannot be achieved by any attempt to improve the vascular supply, is undoubtedly tenotomy of the tendo Achillis. All except two patients have been able to walk more than a mile thereafter and the two who halted at less than that distance did so because of anginal pain. The operation, although trivial in itself, does cause slight disability and certainly will not permit elderly and shuffling patients with claudication to walk with the brisk gait of young men whose pain has been relieved. It does, however, relieve them of the pain which has made their lives a misery and it enables them to spend their remaining days in reasonable activity. For the Type 2 case of claudication, when age and general condition permit, lumbar ganglionectomy must be rated as the most successful treatment. In more elderly patients chemical sympathectomy has its place. Further comment on the results of treatment with α-tocopherol must be withheld until the results of controlled experiments are known. Type 1 cases can be cured by any form of therapy in which the patient has faith. The one failure in this series was a patient who could not be persuaded that his condition was not incurable.

SUMMARY

1. A description is given of historical discoveries relating to intermittent claudication. Various theories that have been advanced are discussed. A hypothesis, based on the work of Lewis, is elaborated.

2. A classification of obliterative arterial disease is outlined. The three groups that are distinguished are: primary thrombosis of the popliteal artery; juvenile obliterative arteritis; and senile obliterative arteritis.

3. The methods adopted for assessment of the severity of disease, including study of the clinical features, arteriographic findings, results of novocain infiltration and examination of the patient on a walking machine, are reported.

4. Methods of treatment by Buerger's exercises, contrast baths, intermittent venous occlusion and suction pressure; by lumbar ganglionectomy and paravertebral block with phenol; by vitamin E (α-tocopherol) therapy; by treatment with thiouracil and antistin; by internal popliteal myoneurectomy and division of the external popliteal and posterior tibial nerves; and by tenotomy of the tendo Achillis, are discussed.

5. It is concluded that tenotomy of the tendo Achillis should replace myoneurectomy in Type 3 cases where the blood supply is so far reduced that vascular stability cannot be achieved, and that it might apply in Type 2 cases in which there is persistent pain at a steady level.

6. The results of treatment in 276 patients with intermittent claudication are recorded.

REFERENCES

BAER, S., HEINE, W. I., and GELFOND, D. B. (1948): American Journal of the Medical Sciences, **215**, 542.
BAER, S., and HEINE, W. I. (1949): Journal of the American Medical Association, **139,** 733.
BARSOUM, G. S., and GADDUM, J. H. (1935): Journal of Physiology, **85,** 13.
BOULEY, P. (1831): Recueil de médecine vétérinaire (*cit.* Lewis), **8,** 517.
BOYD, A. M. (1938): St Bartholomew's Hospital Reports, **71,** 151.
BOYD, A. M., and JEPSON, R. P. (1949): In Press.
BOYD, A. M., and WILDE, F. R. (1949): In Press.
BRODIE, B. (1846): Lectures Illustrative of Various Subjects in Pathology and Surgery. London: Longman, Brown, Green and Longmans, p. 356.
BUERGER, L. (1908): American Journal of Medical Sciences, **136,** 567.
CHARCOT, J. M. C. (1858): Compte rendu hebdomadaire des séances et mémoires de la Société de Biologie, (*cit.* Ejrup), **5,** 225.
CHARCOT, J. M. C. (1887): Progrès médical, 2e serie, **6,** 99.
COMROE, J. H. (1923): Annals of Clinical Medicine, **1,** 313.
EJRUP, B. (1948): Tonoscillography after Exercise. Supplement 211, Acta Medica Scandinavica.
ERB, W. (1898): Deutsche Zeitschrift für Nervenheilkunde, **13,** 1.
HAMILTON, W. F. (1947): Howell's Text-book of Physiology. Philadelphia: Saunders.
HAXTON, H. A. (1947): Personal Communication.
JEPSON, R. P., and RATCLIFFE, A. H. (1949): In Press.
KATZ, L. N., LINDNER, E., and LANDT, H. (1935): Journal of Clinical Investigation, **14,** 807.
KELLGREN, J. H. (1938): Clinical Science, **3,** 175.
KROGH, A. (1922): The Anatomy and Physiology of Capillaries. New Haven: Yale University Press.
LEARY, W. V., and ALLEN, E. V. (1941): American Heart Journal, **22,** 719.
LERICHE, R. (1947): Medical Research in France during the War 1939–45. Editor: J. Hamburger. Paris: Editions Medicales Flammasion, p. 275.
LEWIS, T. (1942): Pain. New York: Macmillan.
LEWIS, T., and KELLGREN, J. H. (1939): Clinical Science, **4,** 47.
LEWIS, T., PICKERING, G. W., and ROTHSCHILD, P. (1929): Heart, **15,** 359.
LIAN, C. (quoted by THOMAS, A., 1922): Les Phènoménes de Répercussivité. Paris: Masson et Cie.
LINDQUIST, T. (quoted by Ejrup: 1948).
MANDL, F. (1948): Paravertebral Block. London: Heinemann.
MARINESCO, G. (1896): Sémaine médicale, **16,** 65.
OGILVIE, H. (1949): Beyer Memorial Lecture. British Medical Journal, **1,** 645.
PEARL, F. L. (1937): American Journal of the Medical Sciences, **194,** 505.
SIMMONS, H. T. (1936): Lancet, **1,** 73.
THOMAS, A. (1922): Presse médicale, **30,** 1049.
VEAL, J. R., and MCFETRIDGE, E. M. (1936): American Journal of Medical Sciences, **192,** 113.
VOGELSANG, A. B., SHUTE, E. V., and SHUTE, W. E. (1947): Medical Record, N.Y. **160,** 21, 91, 163, 230, 279.
VOGELSANG, A., SHUTE, E. V., and SHUTE, W. E. (1948): Medical Record, N.Y. **161,** 83.
WINIWATER, F. V. (1879): Archiv für Klinische Chirurgie, **23,** 202.
ZAK, E. (1921): Wiener Archiv für innere Medizin, **2,** 405 (*cit.* Ejrup).

THE FATE OF VOLUNTARY MUSCLE AFTER VASCULAR INJURY IN MAN

RUTH E. M. BOWDEN, LONDON, ENGLAND

Department of Anatomy, Royal Free Hospital School of Medicine, London; formerly of the Nuffield Department of Orthopaedic Surgery, University of Oxford

and

E. GUTMANN, PRAGUE, CZECHOSLOVAKIA

Department of Neuropathology, Prague; formerly of the Department of Zoology and Comparative Anatomy, University of Oxford

Much has been written about the fate of voluntary muscle that has been partly or wholly deprived of its blood supply, but there is still disagreement as to the pathogenesis of the lesions. Confusion is understandable because ischaemic damage of muscle is encountered in many types of case and on the basis of clinical and experimental findings it has been attributed to many different causes. It is found in open wounds with injury to the main vessels or their muscular branches, in closed injuries of the limbs with damage to the vessels, and in acute embolic catastrophies. Ischaemia of more gradual onset may be due to thrombosis of arteries or veins, or to the accumulation of fluid or the swelling of muscles within a rigidly bound space such as the anterior tibial compartment.

Ischaemic damage of muscle has been ascribed to: arterial occlusion (Volkmann 1878, Griffiths 1940, Barnes and Trueta 1942, Parkes 1945, Cohen 1948); arterial spasm (Leriche 1935, Cohen 1941, 1944, 1948); venous obstruction (Brooks 1922, Brooks and Martin 1923, Brooks, Johnson and Kirtley 1934, Middleton 1930); external pressure alone (Jepson 1926, Lewis 1936); external pressure with ligation of veins (Jepson 1926); capillary paralysis (Leveuf 1937); and peripheral nerve injuries (Putti 1938).

Clearly, as pointed out by Leveuf (1937), Griffiths (1940), and Parkes (1945), conditions of different etiology and pathology have been grouped together solely on the ground that they produce contracture in voluntary muscles. In fact, however, two lesions of strikingly different type have been reported: one is characterised by massive necrosis of contractile tissue; and in the other there is dense intramuscular fibrosis. Classification of these lesions cannot be based satisfactorily on clinical findings alone, but histological classification may serve to distinguish them and shed light on the problems of etiology.

Biopsies of muscle were taken from sixteen patients with vascular lesions treated at the Peripheral Nerve Injury Centre in Oxford. The methods of clinical examination that were

Figures 1–9

All preparations are of human muscle. Unless otherwise stated the material has been stained with a modification of Bielschowsky's technique. FIG. 1—Muscle fibres from a case of early necrosis. The nuclei are still visible, but there is variation in the intensity of the staining reaction in different muscle fibres. FIG. 2—Discoidal fragmentation of muscle fibres in a necrotic area (haematoxylin and eosin). FIG. 3—Cross-section of muscle showing a sharply defined area of necrotic muscle fibres surrounded by a zone of phagocytic and fibroblastic activity. Note the dense acellular fibrosis at the periphery (Mason stain). FIGS. 4–5—Phagocytic activity leading to breakdown of the fibres at the edge of the necrotic mass of muscle (Hortega silver carbonate stain). FIG. 6—Scattered fragments of necrotic muscle fibres within the connective tissue in an area of incomplete devascularisation adjacent to an area of massive necrosis (Mason stain). FIG. 7—Incomplete devascularisation of muscle. Note the ill-defined border-line of vascular connective tissue which is infiltrating between the muscle fibres. Note the wavy outline of these muscle fibres, in some of which there is an apparent increase in the sub-sarcolemmal nuclei. These findings indicate the effect of denervation (Hortega silver carbonate stain). FIG. 8—Showing two adjacent areas of vascular damage which is complete on the right and incomplete on the left. Compare the sharp and ill-defined borders of the two regions (Hortega silver carbonate stain). FIG. 9—Regeneration of muscle fibres in an area of incomplete devascularisation. Note the multinucleated and bulbous ends of the myotubes on the left.

FIG. 1

FIG. 2

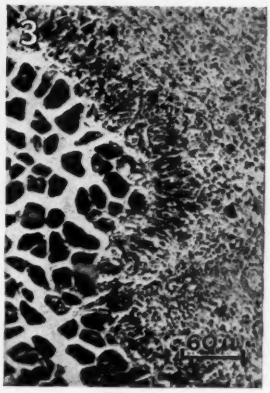


FIG. 3

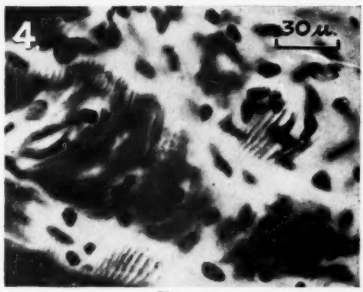


FIG. 4

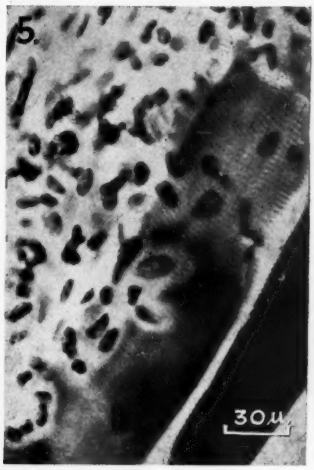


FIG. 5

FIG. 6

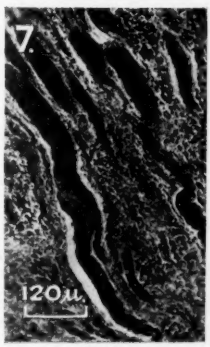


FIG. 7

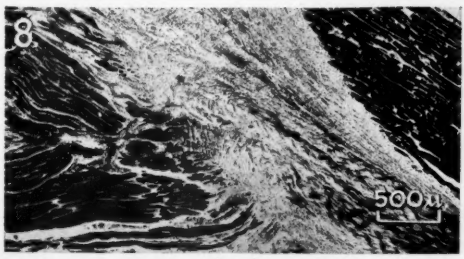


FIG. 8

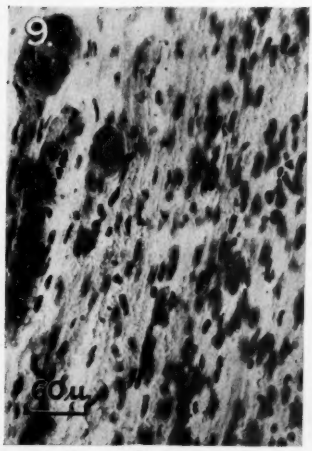


FIG. 9

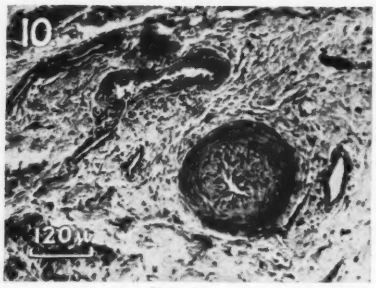


FIG. 10

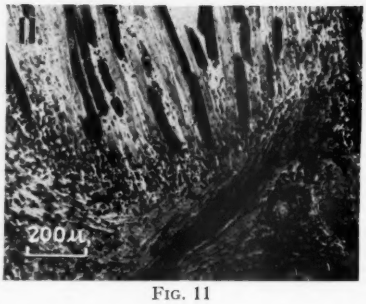


FIG. 11

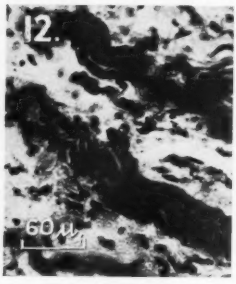


FIG. 12

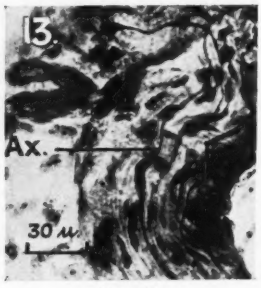


FIG. 13

FIG. 14

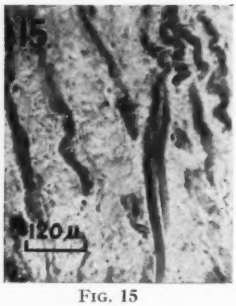


FIG. 15

FIG. 16

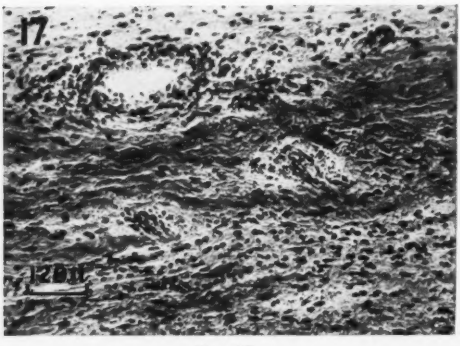


FIG. 17

FIG. 18

used have been reported previously by Holmes, Highet and Seddon (1944); and the method of taking the biopsies, and the histological technique employed, were described by Bowden and Gutmann (1945).

Three types of lesion in muscle—The clinical and histological findings are summarised in Table I. Many observers have described the changes that occur in muscle after vascular damage, but a summary of the findings in this series is relevant. Three main types of lesion could be distinguished histologically. In the first there was massive necrosis of muscle fibres, the necrotic areas being surrounded by a sharply defined zone of fibrous tissue; in the second there was dense interstitial fibrosis; and the third group showed elements of both these types in that there were scattered foci of necrosis together with interstitial fibrosis.

Group 1—*Massive necrosis of muscle*—Biopsy was performed in five patients, at intervals ranging from 40 to 800 days after injury. The muscles were hard and friable with patches of yellow-green necrotic tissue. When muscle fibres could be identified they were arranged in straight lines; some were of normal diameter and some were swollen. There was no evidence of fibrillar structure within the muscle fibres which showed considerable variation in staining-reaction even in a single section (Fig. 1). In longstanding cases no nuclei were seen, but in more recent ones there were a few small nuclei of abnormal appearance and staining-reaction. When cross-striation was present it was unusually clear, although again there was no uniformity of staining (Figs. 1 and 2). Transverse discoidal fragmentation was easily recognised (Figs. 2 and 4). The necrotic areas were surrounded by a clearly defined zone of fibrous tissue but there was complete absence of fibrosis between the necrotic muscle fibres (Fig. 3). In more recent cases the collagen fibres were loosely arranged, enmeshing a few red blood cells. In later ones the fibres were densely packed, especially at the periphery. At the inner margin of this ring of fibrosis there was evidence of activity by macrophages which appeared to be breaking the muscle fibres into fragments, some no larger than a single horizontal band (Figs. 4 and 5). As these remnants of muscle were removed they were apparently replaced by fibrous tissue. Similar changes have been reported by Kraske (1879), Leser (1894), Scott (quoted by Burrows 1918), Bristow (quoting Greenfield 1923), Griffiths (1940), Barnes and Trueta (1942), Le Gros Clark and Blomfield (1945), and others.

In areas adjacent to the site of necrosis there were ill-defined zones of highly vascular connective tissue (Figs. 6–8). The muscle fibres scattered throughout this fibrous tissue showed signs of necrosis (Fig. 6), denervation (Fig. 7) or regeneration (Fig. 9). The richly nucleated and bulbous muscle fibres that were seen in this zone afforded proof of the possibility of recovery of function by the regeneration of contractile tissue.

The endothelium of intramuscular blood vessels showed proliferation but it was impossible to judge whether this endothelial hyperplasia was the cause or the result of the necrosis of muscle (Fig. 10). The intramuscular pattern of innervation was also studied. In five cases in which massive necrosis was found, empty nerve trunks were seen in areas adjacent to the necrosis (Figs. 11 and 12). In specimens from two patients thin axons, resembling regenerated nerve fibres, were seen penetrating the empty Schwann tubes (Fig. 13). In one, the axons and

Figures 10–18

All preparations are of human muscle. Unless otherwise stated the material has been stained with a modification of Bielschowsky's technique. FIG. 10—Showing an artery with narrowed lumen and endothelial proliferation in an area adjacent to a patch of necrosis (haematoxylin and eosin). FIG. 11—Note the empty nerve trunk on the left of a patch of completely necrotic muscle fibres which show marked lack of uniformity in staining reaction. FIG. 12—Empty Schwann tubes lying in connective tissue adjacent to an area of necrosis. FIG. 13—Nerve trunk adjacent to a necrotic area. Note the thin regenerating axons (Ax). FIG. 14—Dense intramuscular fibrosis. Note the muscle fibres scattered in the connective tissue. FIG. 15—Degenerated muscle fibres showing as homogeneous bands within connective tissue in which there are scattered nuclei. FIG. 16—Infiltration of perivascular region by small round cells (haematoxylin and eosin). FIG. 17—Perivascular infiltrations in an area of dense fibrosis. FIG. 18—Fibrosis and small round cell infiltration between muscle fibres which show the effect of denervation.

myelin sheaths appeared to be intact, but close inspection showed that the Schwann cells were devoid of nuclei (*cf.* Holmes, Highet and Seddon 1944)—a condition that is analogous to the necrosis of muscle fibres. This appearance of denervated fibres was described by Leveuf (1937) but he interpreted it, probably on account of the real or apparent increase in the size of nuclei, as evidence of an " acute irritative process " in the muscle itself.

The process of regeneration of muscle was studied in the rabbit by Blomfield and Le Gros Clark (1945) and the appearance of richly nucleated, bulbous muscle fibres found in human cases in this series was identical with those found in their experiments. On the basis of the clinical course of certain cases of ischaemic paralysis Leveuf suggested that regeneration of muscle must take place, although at the time he had no histological evidence of it.

Group 2—*Diffuse interstitial fibrosis*—In the six patients who were studied, biopsies were taken at intervals varying from 95 to 700 days after injury. The muscles were paler than normal and slightly firmer, and the changes were predominantly those of dense interstitial fibrosis. Muscle fibres were scattered throughout the connective tissue and showed a variety of changes: many appeared to have escaped serious damage, nuclei and cross-striation being intact; others showed signs of denervation, and isolated fibres were necrotic (Figs. 14 and 15). There were many extravasations of blood in various stages of organisation, particularly in one specimen where the muscle fibres showed only signs of denervation. Nerve trunks were seen only in two cases: in one there were axons indistinguishable from regenerated fibres; and in the other the Schwann tubes were empty.

Group 3—*Scattered foci of necrosis together with interstitial fibrosis*—This group consisted of five patients. Biopsies were taken at intervals ranging from 126 to 331 days after injury. The muscles were paler than normal; in three, there were scattered foci of yellow-green necrotic tissue varying in size from microscopic patches up to areas 1 centimetre in diameter. Histological examination showed isolated regions of necrotic muscle surrounded by fibrous tissue; some denervated muscle fibres were also seen. The predominant change was that of scattered patches of interstitial fibrosis but, in one case, organised extravasations of blood were conspicuous; and in two, there were perivascular collections of small round cells like those seen in chronic inflammatory lesions (Figs. 16–18). The pattern of innervation varied. No nerve trunks were seen in one specimen; in another there were apparently normal nerve fibres and regenerating axons; and three exhibited regenerating nerve fibres and empty Schwann tubes in an area showing signs of previous vascular disturbance. In one case the appearance was typical of delayed re-innervation, but fibrosis of the muscle was probably due largely to ischaemia because it was far greater than would have been expected in a muscle denervated for less than a year.

DISCUSSION

Relationship of the type of vascular injury to the histological changes—*Group I*—It will be seen from Table I that in the first group, where there was massive necrosis of muscle, there had been serious damage to the main artery of the limb or to the vessel supplying the affected muscles. There was complete obstruction of the main vessel in Cases 2 and 5, and of the branches to the muscles in Case 3. In Cases 1 and 4 the brachial artery, although patent, was constricted and greatly reduced in diameter over a considerable length.

It is well known that constriction or division of a main vessel does not invariably lead to massive necrosis (Cases 9 and 14 of this series being examples). Indeed Griffiths (1940) and Barnes and Trueta (1942) encountered great difficulty when attempting to produce massive necrosis of muscle in the rabbit. Leriche, Cohen and others suggested that the arterial spasm must extend far enough to involve the vessels on which the collateral circulation depends; if spasm of the main vessel involved the collaterals, or the branches supplying the muscles, and if it persisted for a sufficient time, massive necrosis occurred.

Lewis (1936) stated that deprivation of arterial blood supply for six to ten hours resulted in death of the muscle. The extensive constriction of the artery found in Case 4 is of interest. Spasm of arteries resulting from injury is well recognised, and Belsey (1941) reported a case in which the femoral artery was seen to be in spasm five days after a crush injury of nine hours' duration. It is not proved that prolonged spasm can lead directly to fibrosis of the vessel, but it is tempting to suggest that this may have been so in Cases 9 and 14.

Albert and Mitchell (1943) considered that necrosis might be due either to temporary arterial obstruction or to venous occlusion. Damage confined to the arteries supplying muscles led to necrosis in the cases reported by Parkes (1945), Power (1945), and in Case 3 in this series. Cohen (1948) reported massive necrosis of the thenar muscles after thrombosis of the radial artery due to accidental intra-arterial injection of pentothal. Massive necrosis has also been reported after damage to the veins. In Case 9 reported by Parkes the femoral vein had been ligated for secondary haemorrhage after an operation for varicose veins and the histological picture of the muscles differed in no way from those of the first group reported here. There was no evidence of damage to the femoral artery, but in view of the close proximity of this vessel to the femoral vein the possibility of temporary spasm due to the operative interference cannot be excluded.

Group 2—When there was dense interstitial fibrosis, damage to the main vessels was more varied. In Cases 6 and 7 trivial injury to the limb had caused slow haemorrhage into the rigidly bound anterior tibial compartment. In Case 8 there was a simple through-and-through gunshot wound of the arm; no damage to the vessels was detected at operation 150 days later, but there was marked gravitational cyanosis distal to the level of injury. In Case 9 the brachial artery and vein had been severed and there was marked oedema of the limb. In Case 10 the femoral vein was damaged by a gunshot wound and had been ligated. In Case 11 there was ischaemic necrosis of the median nerve due to injury to its main arterial supply (Seddon and Holmes 1945) but there was no definite evidence of injury to the muscular branches of the artery; a tight plaster might have accounted for the ischaemic damage to the muscles.

Group 3—In this group, where there was patchy fibrosis with discrete islands of necrosis, there was again no uniformity in the causal factors. In Case 12, trivial injury to a patient who had a low Vitamin C level in the plasma caused slow haemorrhage into the anterior tibial compartment. Case 13 received an extravascular transfusion of blood (not less than one and not more than two pints) into the anterior tibial compartment within an hour of sustaining multiple injuries. Case 14, a boy aged ten years, with a closed supracondylar fracture of the humerus, had signs of acute contracture within an hour of injury. At operation, 180 days after injury when the specimen of muscle was taken, the brachial artery was found to be constricted; the median and ulnar nerves appeared to be normal. In Case 15 the patient sustained a closed fracture of the lower third of the tibia and fibula. There was gross swelling of the limb but no evidence of direct injury to the vessels. A tight plaster may have contributed to the onset of the ischaemic lesion.

Similar patchy necrosis has been reported in muscles after crush injuries (Bywaters and Beall 1941, and Belsey 1941). The factors that might have contributed to these changes in the muscle were severe local damage to the tissue, traumatic spasm of the main arteries, or accumulation of fluid under pressure beneath the deep fascia. In two of Belsey's cases the main artery of the limb was found in spasm; in five of them, the average duration of crushing was eight hours. He reports one, however, where the patient was trapped under debris for only forty-five minutes. As Belsey points out, this alone does not give any indication of the duration of arterial spasm. Local swelling was found at the site of the crush and in Cases 2 and 3 of his series there was intense swelling of the limbs. When the deep fascia was incised a considerable amount of fluid was released. In one case reported by Bywaters and Beall the limb became pulseless although there was no progressive swelling of the limb.

TABLE I

Case	Age in years	Nature of Injury Associated lesions and abnormalities	Time in days between injury and biopsy	Clinical Evidence of Circulatory Disturbance			Functional Tests of Muscles						
				At time of biopsy			Muscles			Electrical reactions		Electromyography	
				Approximate time of onset	Pulse	Oscillometry	Arteriogram	Consistency	Contracture	Voluntary power (footnote 1)	Faradism (footnote 2)	Galvanism (footnote 2)	Motor unit action potentials	Fibrillation action potentials
Group I														
1 B.178	8	Supracondylar fracture humerus. Median, ulnar and incomplete radial palsy. ? Tight plaster	40	1 hour	Absent	—	—	Flexors hard Intrinsics soft	+++ 0	0 0	— 0	— S	— —	— —
2 S.35	37	Colles' fracture. Ischaemic nerve lesions. Tight plaster	120	12 hours	Good	Diminished lower half forearm	Obstruction radial artery at level of fracture	Hard in hand and lower half forearm	+++	4 Hand 0	0 0	0 S	—	—
3 G.58	27	Fracture radius and ulna. Ischaemic lesions median and ulnar nerves. Tight plaster	180	24 hours	Good	No abnormality detected	Narrowing of brachial artery. Absent muscular branches. Break in anterior interosseous artery	Firm flexors Intrinsics soft	++ 0	0 0	0 0	0 S	—	—
4 B.72	20	Dislocation of elbow. Median, ulnar and radial nerve lesions. Splinted with acute flexion of elbow. Severe burn later	221	24 hours	Weak	—	Constriction of brachial artery	Firm below elbow	+++	Forearm 1 Hand 0	0 0	0 S High	— Silent	— 0-1
5 G.24	60	Multiple fractures. Complete brachial plexus lesion. Severe bruising upper limb	800	20 hours	Absent	Absent	Complete obstruction of the brachial artery at the level of neck of humerus	Firm	++	Prox. 2–3 Dist. 0	+ 0	+ 0	— 0	— 0
Group II														
6 J.43	29	None known. Plasma vitamin C 0·03 mg./100 cc. (Normal 0·3)	95	2–3 days	Slightly diminished	Slightly diminished	No lesion seen	Firm	Extensor hallucis longus only	2 Extensor digitorum longus 0	0 0	+ 0	— —	— —
7 A.30	20	Pain after kicking ball. Incomplete lateral popliteal nerve injury. Haematoma	106	6–12 hours	Good	Slightly increased	Technically unsatisfactory	Firm	+	3	0	0	+	+
8 P.57	26	Shell wound. Incomplete division of ulnar nerve. Sepsis	150	No note	Gravitational cyanosis	—	—	Soft	0	Ulnar innervation 0 Median 5	0	S	—	—

FOOTNOTES TO TABLE I

1. Voluntary Power—
   0 = paralysed.
   1 = flicker.
   2 = movement of joint with gravity eliminated.
   3 = movement against gravity.
   4 = movement against resistance, but weaker than normal.
   5 = normal.

2. Electrical Reactions—Percutaneous faradic and galvanic stimulation—
   0 = no reaction.
   + = response.
   S = sluggish contraction.
   High = abnormally high threshold stimulus.

3. Histology—Nerve fibres—
   0 = None seen.
   1 = Few thin nerve fibres seen, many empty Schwann tubes. No innervated end-plates.
   2 = Nearly all nerve trunks innervated, mostly with thin nerve fibres, a few thicker myelinated fibres and innervated end-plates.
   3 = All nerve trunks innervated, axons thick but diameter and myelination not yet normal.
   4 = Normal pattern and degree of innervation.

TABLE I

Sensory and Sudomotor Changes	Operative Findings			Histology (footnote 3)			Clinical Progress (footnotes 4 and 5)
	Muscles	Vessels	Nerves	Muscle	Nerve fibres	Prognosis	
Glove anaesthesia and analgesia. Anhidrosis except in interdigital clefts	Lower half of flexors necrotic	Brachial artery 2 mm. in diameter over a length of 8 cms., feeble pulse	Ulnar slightly indurated and swollen Median acute angulation over fracture, slightly indurated	Necrosis predominant Some regeneration of muscle fibres	1	C	Radial M_4 S_2 Median M_4 S_2 Ulnar M_3 S_2 Three months after injury (No further information available)
Glove anaesthesia, analgesia and anhidrosis	Severe, patchy necrosis	Radial pulse absent at level of fracture	Median and ulnar fibrotic and shrunken, due to ischaemia (footnote 6)	Necrosis predominant Foci of fibrosis with denervated fibres	—	C	Radial M_3 Median $M_1(_5)$ S_1 Ulnar M_2 S_3 Six years after injury
Glove anaesthesia and analgesia. Sweating diminished, particularly in the periphery	Hard, friable, necrotic	Muscle branches and anterior interosseous artery fibrous cords only	Shrunken and fibrotic for 7 cms. Ischaemic lesion	Necrosis with regeneration of muscle fibres	1	B	M_5 S_4 Two years after injury
Glove anaesthesia, analgesia and anhidrosis	Friable, necrotic, thick capsule	Brachial artery constricted for 4 cms.	Fibrotic	Necrotic	0	C	Radial M_4 S_1 Median M_2 S_1 Ulnar M_2 S_0 Five years after injury
Gross hypoaesthesia and hypoalgesia. Glove anhidrosis	Necrotic	—	Median and ulnar fibrotic, shrunken. Ischaemic (footnote 7)	Necrosis and fibrosis of muscle	—	C	Median M_1 S_1+ Ulnar M_1 S_1+ Five years after injury
Anaesthesia first toe and interdigital cleft. Analgesia less extensive. Slight hyperhidrosis under test.	Pale	—	—	Dense fibrosis. No necrosis of muscle fibres	—	C	M_3 S_4 Two years after injury
Incomplete lateral popliteal anaesthesia. No analgesia or sweating change	Pale, not hard	Fragile	Normal	Early collagenisation Many extravasations No necrosis of muscle fibres	—	C	M_3 S_4 Four years after injury
Ulnar anaesthesia and analgesia; incomplete anhidrosis in distribution of the ulnar nerve	Slightly pale	No abnormality detected	Ulnar nerve gross scarring. Resection and suture necessary	F.C.U. fibrosis. Ruptured veins. Extravasation of blood. Moderately severe denervation atrophy	1-2	B	M_1 S_2+ Four years after injury

FOOTNOTES TO TABLE I

4. Recovery of motor power—

M_0 = Absence of contraction.
M_1 = Return of perceptible contraction in the proximal muscle. Figures in parenthesis after this indicate strength of muscles.
M_2 = Return of perceptible contraction in the proximal and distal muscles.
M_3 = Recovery of function in important distal muscles sufficiently strong to overcome resistance.
M_4 = Recovery of function as in 3, but in addition there is recovery of independent and synergic movement.
M_5 = Complete recovery.

5. Recovery of sensation—

S_0 = Absence of sensibility in the autonomous zone.
S_1 = Recovery of deep pain sensibility in the autonomous zone.
S_2 = Partial recovery of superficial touch and pain.
S_3 = Complete recovery of touch and pain without any abnormal response.
S_4 = Recovery as in 3, but with additional recovery of two point discrimination.

6. Case 5 of Holmes, Highet and Seddon (1944).

7. Case 4 of Holmes, Highet and Seddon (1944).

TABLE I

Case	Age in years	Nature of Injury Associated lesions and abnormalities	Time in days between injury and biopsy	Clinical Evidence of Circulatory Disturbance				Functional Tests of Muscles						
				Approximate time of onset	At time of biopsy		Arteriogram	Muscles			Electrical reactions		Electromyography	
					Pulse	Oscillometry		Consistency	Contracture	Voluntary power (footnote 1)	Faradism (footnote 2)	Galvanism	Motor unit action potentials	Fibrillation potentials
9 W.119	33	Multiple shell wounds. Compound fracture humerus. Division of brachial artery, vein and median nerve. Incomplete division of ulnar nerve. Gross sepsis	178	+	Absent	—	—	Firm	+++ —	0 0	0 Oedema 0	0 S	— Silent	— Feeble
10 M.55A	30	Multiple shell wounds. Division of femoral vein	210	No note	Absent	—	Slight narrowing of femoral artery	Firm	+	0 0	0 0	0 0	Silent 1	Silent 0
11 B.94	28	Compound fracture of radius and ulna. Median and ? radial nerve injury. Tight plaster	700	No note	Radial absent Ulnar present	—	—	Slightly firm	+	In muscles examined histologically 0	High	S	1–2	Silent
Group III 12 S.110	40	Stubbed toe when playing football. Plasma vitamin C 0·02 mgm. %	126	4 days	Good	Increased	Lumen narrowed as vessel passed into anterior tibial compartment	Hard	0	0 2	0 0	0 0	— —	— —
13 W.121	23	Accidental intramuscular transfusion	150	No note	Good	Both legs damaged	—	Hard	+	0	0	0	Silent	Silent
14 L.24	10	Supracondylar fracture humerus. Median, ulnar and incomplete radial nerve injuries	180	½ hour	Weak	Diminished	Constriction of brachial artery	Firm proximally, soft distally	+ 0	1–2 0	+ 0	+ S	— —	— —
15 H.64	21	Fracture R. tibia and fibula lower third. Gross swelling ? Tight plaster, second to ninth day. Skeletal traction applied tenth day. Sciatic palsy noted three weeks after injury	210–240	+	Good	Diminished	—	Soft	—	FLH, ELH, EDB 2–3 Intrinsics 0	Oedema		+ patchy	1-2
16 M.65	30	Multiple shell wounds. Compound fracture humerus and elbow. Severe incomplete lesions of radial, median and ulnar nerves. Division radial and ulnar arteries. Sepsis	331	+	Absent	—	—	Hard	+++	0	0	0	—	—

FOOTNOTES TO TABLE I

1. Voluntary Power—
   - 0 = paralysed.
   - 1 = flicker.
   - 2 = movement of joint with gravity eliminated.
   - 3 = movement against gravity.
   - 4 = movement against resistance, but weaker than normal.
   - 5 = normal.

2. Electrical Reactions—Percutaneous faradic and galvanic stimulation—
   - 0 = no reaction.
   - + = response.
   - S = sluggish contraction.
   - High = abnormally high threshold stimulus.

3. Histology—Nerve fibres—
   - 0 = None seen.
   - 1 = Few thin nerve fibres seen, many empty Schwann tubes. No innervated end-plates.
   - 2 = Nearly all nerve trunks innervated, mostly with thin nerve fibres, a few thicker myelinated fibres and innervated end-plates.
   - 3 = All nerve trunks innervated, axons thick but diameter and myelination not yet normal.
   - 4 = Normal pattern and degree of innervation.

TABLE I

Sensory and Sudomotor Changes	Operative Findings			Histology (footnote 3)			Clinical Progress (footnotes 4 and 5)
	Muscles	Vessels	Nerves	Muscle	Nerve fibres	Prognosis	
Median and ulnar anaesthesia and analgesia. Glove anhidrosis	Pale	—	—	Dense fibrosis Intramuscular extravasations of blood	0	C–B	Median $M_1+ S_0$ Ulnar $M_1 S_1+$ Four years after injury
Complete sciatic anaesthesia and analgesia. Complete lateral popliteal and incomplete medial popliteal anhidrosis	Pale	No abnormality detected	No abnormality detected	Dense fibrosis, scattered extravasations of blood. Mixed degeneration and denervation of muscle fibres	—	B	$M_3 S_3$ Three years after injury
Median and superficial radial sensory loss. Glove anhidrosis	Pale	Venous engorgement	10 cms. threadlike grey, avascular trunk. Peripheral stump vascular but shrunken	Dense fibrosis. No necrosis of muscle but atrophy of denervation. Scattered extravasations of blood	—	C–B	No change Repair of nerve impossible
None	Tibialis anterior necrotic. Extensor digitorum longus yellow	—	—	Dense fibrosis. Extravasation and perivascular infiltration. Arteries thick. Foci of necrotic muscle fibres	—	C	$M_1+(3\frac{1}{2})\ S_4$ Three years after injury
Lateral popliteal anaesthesia, analgesia and anhidrosis	Yellow-green necrotic	—	—	Dense fibrosis. Extravasations, perivascular infiltrations. Scattered foci of necrotic muscle fibres	0–1	D	Lat. pop. $M_1 S_1+$ Four years after injury
Glove hyperaesthesia and hyperalgesia. Patchy anhidrosis	Prox. muscle pale. Distal half of flexors patchy necrosis	Brachial artery 2 mm. external diameter	Median and ulnar normal	PRT minor degree vascular lesion. Patchy dense fibrosis small areas necrosis. Atrophy of denervation	4 0–2	B	$M_4 S_4$ Patient died five years after injury
Sciatic anaesthesia, analgesia and anhidrosis	Pale	—	—	Early collagenisation, patchy necrosis with atrophy of denervation	1–2	B	$M_5 S_4$ Two years after injury
Patchy areas of anaesthesia, analgesia and anhidrosis in glove distribution	Pale and wasted	—	—	Dense fibrosis, many small collagenised extravasations of blood. Scattered necrotic muscle fibres	0–1	C	Radial M_0 S— Median $M_1 S_2$ Ulnar $M_0 S_0$ Five years after injury

FOOTNOTES TO TABLE I

4. Recovery of motor power—

M_0 = Absence of contraction.

M_1 = Return of perceptible contraction in the proximal muscle. Figures in parenthesis after this indicate strength of muscles.

M_2 = Return of perceptible contraction in the proximal and distal muscles.

M_3 = Recovery of function in important distal muscles sufficiently strong to overcome resistance.

M_4 = Recovery of function as in 3, but in addition there is recovery of independent and synergic movement.

M_5 = Complete recovery.

5. Recovery of sensation—

S_0 = Absence of sensibility in the autonomous zone.

S_1 = Recovery of deep pain sensibility in the autonomous zone.

S_2 = Partial recovery of superficial touch and pain.

S_3 = Complete recovery of touch and pain without any abnormal response.

S_4 = Recovery as in 3, but with additional recovery of two point discrimination.

6. Case 5 of Holmes, Highet and Seddon (1944).

7. Case 4 of Holmes, Highet and Seddon (1944).

When there is accumulation of blood or exudate beneath deep fascia, particularly in a rigidly bound space such as the anterior tibio-fibular compartment, the presence or absence of necrosis in the muscles must depend upon the degree of pressure achieved. If this becomes sufficient to occlude the arteries and arterioles, or to initiate arterial spasm, necrosis might ensue; whereas if the venous return alone is impeded there might be interstitial oedema with subsequent fibrosis. It is implicit in this hypothesis that tension within the tissues of a closed compartment may exceed arterial pressure. Such pressure can be developed only by increase in the effective osmotic pressure of the extravascular tissue fluids, but unfortunately there are no records of measurement of the subfascial pressure in reported cases. It is not difficult to envisage circulatory embarrassment when there are large accumulations of fluid under pressure, but it is less easy to see how slow and uncomplicated intramuscular haemorrhage can lead to necrosis of muscle as in Cases 6 and 7. The etiology of these lesions is not fully understood.

The series of cases reported here supports the concept put forward by Parkes (1945): "On the whole the evidence seems to be in favour of Griffith's contention that true Volkmann's ischaemia with massive necrosis of muscle fibres and their subsequent replacement by fibrous tissue is due to the deprivation of arterial blood supply generally as the result of traumatic arterial spasm. Nevertheless, there is little doubt that a form of muscle contracture clinically resembling the true Volkmann type, though probably without the typical histological picture, can occur from venous or capillary occlusion."

Significance of the intramuscular vascular pattern—While the duration and extent of vascular disturbance are of primary importance, other points demand consideration. The well-known susceptibility of certain muscles such as the flexors of the forearm, and the relative immunity of the forearm extensors and intrinsic muscles of the hand, may be due to several factors. As Parkes (1945) points out, these ischaemic lesions might be caused by arterial spasm of the branches to the flexor muscles, or to quantitatively differing metabolic demands in the two groups of muscle. The work of Campbell and Pennefather (1919), Power (1945), Blomfield and Le Gros Clark (1945), and Blomfield (1945) suggests that there is yet another factor, namely the intramuscular vascular pattern in the individual muscles.

Le Gros Clark and Blomfield investigated the efficiency of intramuscular anastomoses in the leg muscles of the rabbit. They showed that in any particular muscle the vascular pattern was relatively constant. By tying off part of the vascular supply to a muscle, or all of it, they could induce necrosis. The extent of the area affected depended on the site of the vascular damage and the efficiency of the anastomosis. Provided that the anastomosis was adequate, the necrotic area, which was first replaced by fibrous tissue, was later invaded from the periphery by regenerating muscle fibres. It is clear that extensive fibrosis might hinder the growth of these regenerating fibres.

Blomfield, examining human material, found that there were five main types of intramuscular vascular pattern. In the first, there was a longitudinal anastomotic chain entering the muscle throughout most of its length; soleus and peroneus longus showed this arrangement of vessels. The second type was one in which a longitudinal pattern of vessels was derived from a single group of arteries arising from a common stem and entering the muscle at one end, as in gastrocnemius—a muscle which is notoriously susceptible to ischaemia and therefore to clostridial infection. In the third group, to which biceps brachii belongs, there were radiating vessels arising from a single nutrient artery which entered the middle part of the muscle. In the fourth group there was a series of anastomotic loops throughout the length of the muscle; the vessels were derived from a succession of arteries entering the muscle at different levels. Tibialis anterior, extensor hallucis longus, and the long flexors of the toes showed this type of vascular supply. Lastly, there was a group in which sparse anastomoses were arranged in an open quadrilateral pattern. Blomfield concluded that the vulnerability of a muscle to vascular injury was determined by the relation of the

site of injury to the nutrient vessels, the number of the nutrient vessels, the efficiency of the intramuscular anastomoses, the vascular pattern, and the relation between the volume of the muscle and the size of the main nutrient vessels.

Prognosis—The prognosis after vascular injury depends upon the relative size of the masses of viable and non-viable tissue, the possibility of regeneration of muscle, and the state of the nerve supply of the muscle. The muscle injury may be associated with mechanical or ischaemic damage to the main nerve trunk or to the intramuscular nerves. The additional problem of re-innervation therefore arises, though if the greater part of the muscle has been damaged irreparably, even the most complete degree of re-innervation would be useless. Dense interstitial fibrosis is probably irreversible, and recovery after massive necrosis of muscle can be the result only of regeneration of the remaining viable muscle fibres, or of recovery of previously denervated fibres.

SUMMARY

1. Biopsies of muscle were taken during the course of operation from sixteen patients with vascular injuries to the limbs. Three types of histological change were found.

2. In the first, there was massive necrosis of muscle fibres—a group of cases in which there had always been serious damage to the main artery of the limb or to the vessel supplying the affected muscles.

3. In the second type there was dense interstitial fibrosis, the muscle fibres sometimes being normal and sometimes showing necrosis or denervation—a group of cases in which the vascular injury varied from severance of the vessels by gunshot wounds to trivial damage, causing slow haemorrhage within fascial-bound spaces.

4. The third type showed scattered foci of necrosis together with patchy interstitial fibrosis —due to the pressure of tight plasters, crushing of the limb, fractures with arterial contusion, or slow haemorrhage or extravascular transfusion within fascial planes. The rise of tension within the muscles was probably sufficient to occlude the smaller arterioles with resultant patchy necrosis.

5. The vulnerability of certain muscles to vascular damage is partly related to the intramuscular vascular pattern, of which five types have been described.

6. In ischaemic muscles the intramuscular nerve trunks may be normal or they may show evidence of degeneration or necrosis; but in favourable circumstances there may be regeneration of axons.

7. In some cases there was evidence of regeneration of muscle fibres in man, the regeneration being dependent to some extent upon the efficiency of intramuscular anastomoses.

8. The prognosis, in cases of ischaemia of human voluntary muscle, depends upon the extent and the reversibility of damage to both muscle and nerve fibres and upon the extent of regeneration of muscle fibres.

Grateful acknowledgment is made to Mr H. J. Seddon, Professor Lucas Keene, and Professor J. Z. Young, for reading the manuscript, and for their encouragement and criticisms.

REFERENCES

ALBERT, M., and MITCHELL, W. R. D. (1943): Lancet **1,** 519.
BARNES, J. M., and TRUETA, J. (1942): British Journal of Surgery, **30,** 74.
BELSEY, R. (1941): Proceedings of the Royal Society of Medicine, **35,** 328.
BLACKWOOD, W. (1944): British Journal of Surgery, **31,** 329.
BLOMFIELD, L. (1945): Proceedings of the Royal Society of Medicine, **38,** 617.
BOWDEN, R. E. M., and GUTMANN, E. (1945): Lancet, **2,** 768.
BRISTOW, W. R. (1923): British Journal of Surgery, **10,** 478.
BROOKS, B. (1922): Archives of Surgery, **5,** 188.
BROOKS, B., and MARTIN, K. A. (1923): Journal of the American Medical Association, **80,** 1678.
BROOKS, B., JOHNSON, G. S., and KIRTLEY, J. A. (1934): Surgery, Gynecology and Obstetrics, **59,** 496.

BURROWS, H. (1918): British Medical Journal, **1,** 199.
BYWATERS, E. G. L. (1944): Journal of the American Medical Association, **124,** 1103.
BYWATERS, E. G. L., and BEALL, D. (1941): British Medical Journal, **1,** 427.
CAMPBELL, J., and PENNEFATHER, C. M. (1919): Lancet, **1,** 294.
CLARK, W. E. LE GROS (1946): Journal of Anatomy, **80,** 24.
CLARK, W. E. LE GROS, and BLOMFIELD, L. (1945): Journal of Anatomy, **79,** 15.
COHEN, S. M. (1941): Guy's Hospital Gazette, **90,** 201.
COHEN, S. M. (1944): Lancet, **1,** 1.
COHEN, S. M. (1948): Lancet, **2,** 361 and 409.
GRIFFITHS, D. LL. (1940): British Journal of Surgery, **28,** 239.
HOLMES, W., HIGHET, W. B., and SEDDON, H. J. (1944): British Journal of Surgery, **32,** 259.
JEPSON, P. N. (1926): Annals of Surgery, **84,** 785.
JONES, R. (1928): British Medical Journal, **2,** 639.
KRASKE, P. (1879): Centrablatt für Chirurgie, **2,** 193. Quoted by PAGE, H. W. (1900): Lancet, **1,** 83.
LERICHE, R. (1935): Bulletins et Mémoires de la Société Nationale de Chirurgie, **61,** 295.
LESER, E. (1894): In Volkmann's Sammlung Klinischer Vorträge (Chirurgie No. 77), 2087.
LEVEUF, J. (1937): Mémoires de l'Academie de Chirurgie, **63,** 1312.
LEVEUF, J. (1938): Journal de Chirurgie, **51,** 177.
LEWIS, T. (1936): Vascular Disorders of the Limbs. London: Macmillan & Co., Ltd., p. 37.
MIDDLETON, D. S. (1930): British Journal of Surgery, **18,** 188.
PARKES, A. R. (1945): British Journal of Surgery, **32,** 403.
POWER, R. WOOD (1945): British Medical Journal, **1,** 656.
PUTTI, V. (1938): Journal Internationale de Chirurgie, **3,** 189.
SEDDON, H. J., and HOLMES, W. (1945): British Journal of Surgery, **32,** 389.
VOLKMANN, R. VON (1878): Handbuch der allgemeinen und speciellen Chirurgie. Pitha and Billroth. Stuttgart: F. Enke, Band **2,** Abt. **2,** 846.
VOLKMANN, R. VON (1893): Beiträge zur pathologischen Anatomie und zur allgemeinen Pathologie.

SOLITARY PLASMOCYTOMA OF THE SPINE

RONALD W. RAVEN and R. A. WILLIS, LONDON, ENGLAND

From the Royal Cancer Hospital, London

The rarity of solitary plasmocytoma of bone justifies publication of this case of a solitary plasmocytoma of the spine. The opportunity is taken to review authentic cases of the disease in the literature.

CASE REPORT

A male aged fifty-six years, tent erector, was admitted to the Royal Cancer Hospital on October 15, 1947, with paraplegia.

Clinical history—On September 1, 1947, he was lifting a heavy weight and felt something give in his back. One and a half hours later he suffered abdominal pain. On September 10, 1947, he was admitted to another hospital on account of girdle pains. He noticed that his toes were numb, and later they became paralysed. The paralysis extended upwards as far as the level of the umbilicus.

FIG. 1

FIG. 2

Solitary plasmocytoma of the spine. Radiographs of the mid-dorsal region showing forward subluxation of the sixth dorsal vertebra and extensive destruction of two vertebral bodies.

Clinical examination—The general condition was good. No gross abnormality was detected in the heart and lungs. Apart from the paralysis, the abdomen was normal. Girdle pain and hyperaesthesia were present along the distribution of the sixth dorsal segment. Below this level there was paraplegia and anaesthesia. There was incontinence of urine.

Investigations—*Radiographic examination of bones* (Figs. 1 and 2)—*Spine*—Forward subluxation of the sixth dorsal vertebra with incomplete destruction of two bodies—appearances suggestive of a secondary deposit from a tumour. The rest of the spine showed no abnormality. Radiographs of the skull,

pelvis, femora and humeri showed normal appearances. *Urine*—Trace of albumin; moderate number of pus cells with B. coli and B. proteus. *Blood*—Red blood cells, 4,320,000 per c.mm. Haemoglobin, 84 per cent. White blood cells, 18,000 per c.mm. *Differential leucocyte count*—Polymorphs, 75 per cent.; lymphocytes, 20 per cent.; monocytes, 4 per cent.; eosinophils, 1 per cent.; basophils, 0 per cent. *Plasma chlorides*—341 mgm. per 100 c.c. as chlorine, or 562·7 mgm. per cent. as sodium chloride. *Plasma proteins*—6·75 gm. per 100 c.c.; albumin, 4·4 gm. per 100 c.c.; globulins, 1·95 gm. per 100 c.c.; fibrinogen, 0·4 gm. per 100 c.c. *Blood urea*—42 mgm. per cent. *Serum phosphatase*—acid phosphatase, 1·5 units per 100 c.c.; alkaline phosphatase, 6 units per 100 c.c. *Sternal marrow*—Smears showed a normal histological picture; no evidence of a deposit of myeloma. *Erythrocyte sedimentation rate*—21 mm. in one hour (Wintrobe).

Operation—October 23, 1947. Laminectomy was performed with removal of the laminae of the sixth, seventh and eighth dorsal vertebrae. A soft, friable, vascular neoplasm was found eroding the laminae and extending into the erector spinae muscles. The neoplasm surrounded the dura mater but did not involve its posterior aspect. The spinal cord was seen to pulsate. Part of the neoplasm was removed

FIG. 3

Part of the thoracic spine seen from the front. There is collapse of the vertebral bodies and extension of the growth over the heads of the ribs and intercostal spaces.

for microscopical study and the wound was closed. A plaster support was applied to the spine. *Microscopic examination of tissue removed*—The tissue consisted of compact masses of characteristic plasma cells which gave a clearly defined and typical reaction with Unna-Pappenheim methyl green pyronin. The tumour was a plasmocytoma.

Subsequent history—The general health continued to be satisfactory for several months. The urinary bladder was drained continuously with an indwelling urethral catheter until reflex micturition was established six weeks after operation. There was complete anaesthesia with paraplegia below the level of the seventh and eighth dorsal vertebrae, and five weeks after operation increasing involuntary movements developed in the legs. These were partly controlled by luminal and codeine. The patient also complained of severe girdle pains. In March 1948 his general health deteriorated and he died on April 10, 1948.

Necropsy—*Viscera*—The brain, meninges, tongue, tonsils, nasal cavities, pharynx, larynx, trachea, thyroid, heart, oesophagus, stomach, intestines, liver, pancreas, spleen, adrenals, kidneys, testes and the lymph nodes throughout the body showed no significant abnormalities. The lungs showed severe purulent bronchitis and bronchopneumonia with overlying pleurisy of the lower lobes. The bladder showed a moderate degree of cystitis. Extensive sloughing bedsores were present over the sacral region and buttocks. *Skeleton*—The bodies of the sixth and seventh dorsal vertebrae were completely replaced by soft, grey-pink growth with collapse of the bone and free lateral mobility of the spine at this level (Figs. 3 and 4). The intervertebral disc between the two vertebrae was largely intact and lay loosely isolated in the centre of the tumour. The growth had spread anteriorly and laterally beneath the anterior common ligament and over the ribs and intercostal spaces for a distance of 3·5 centimetres on both sides of the vertebrae. It had narrowed the spinal canal and compressed the cord, but had not penetrated through the dura mater. Posteriorly there was invasion of the dorsal spinal muscles especially on the right side. No other tumours were found after careful search in other vertebrae, ribs, sternum, skull, pelvis, and shaft of right femur,

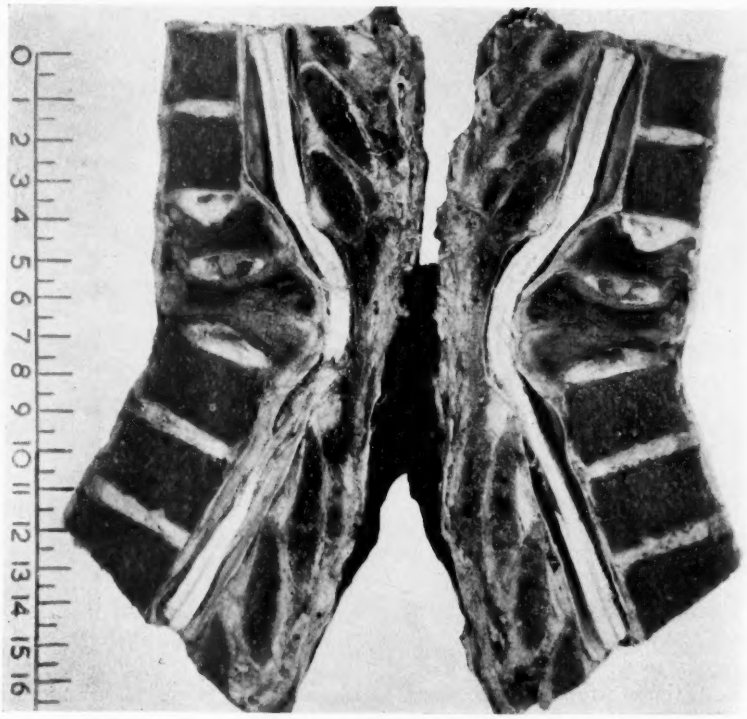


FIG. 4

Median section of the spine shows collapse of the affected vertebral bodies which had been replaced by tumour tissue. Note compression of the cord and isolation of the intervertebral discs.

all of which were sectioned. The shaft of the femur contained red marrow from which smears were taken. *Histo-pathology*—Smears and sections of the vertebral tumour, stained by the usual haematoxylin methods and by the Unna-Pappenheim method for plasma cells, showed it to be a typical plasmocytoma with many cells of poorly differentiated, immature type but also many well-differentiated plasma cells with characteristic structure and staining properties (Fig. 5). The cells showed a moderate number of mitotic figures. A few cells were abnormally large and contained large irregular or multiple (usually only two or three) nuclei. Stromal tissue was scanty. Small blood vessels were plentiful in some parts and there were areas of haemorrhage and degeneration, especially in the central regions of the tumour. Sections and smears of the red marrow from other bones showed no evidence of plasma-cell infiltration. In sections of the kidney, a few tubules contained some brownish amorphous debris, probably of no special significance.

DISCUSSION

Clinical aspects—The first symptoms of solitary plasmocytoma of bone may arise in consequence of pathological fracture after trivial injury, or pain may occur at the site of the lesion. The pain is often severe and, when the lesion is situated in the spine, there may be girdle pain. Paraplegia with all its accompanying features is a complication of solitary plasmocytoma of the spine. General symptoms occur later and include weakness with secondary anaemia.

The radiographic appearances may be grouped into two distinct types. In one type there is a markedly destructive lesion involving the bone, the process being well demarcated and sharply defined. In some respects it resembles a carcinomatous metastasis of the osteolytic variety or the osteolytic type of osteogenic sarcoma. The other type has a cystic and trabeculated appearance, the trabeculae being thickened and irregular. The lesion is large, situated in the medulla of the bone, often expanding the cortex.

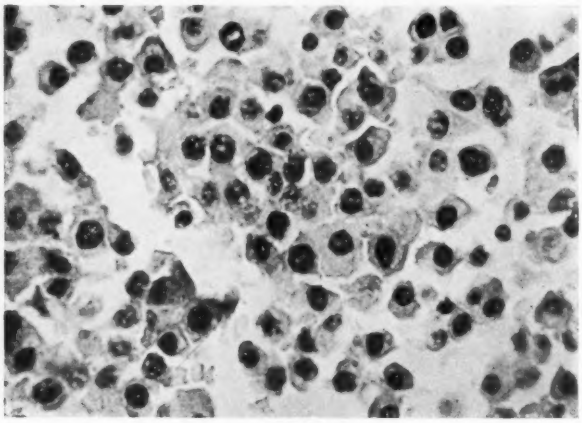


FIG. 5

Photomicrograph of section of tumour (×500).

These solitary tumours are known to be radiosensitive and after high voltage X-irradiation there may be regression with recalcification. When the tumour is situated in a site such as the spine this is the best method of treatment; pain is relieved even after vertebral collapse with paraplegia. Nevertheless, although irradiation may control the disease for a time there is no evidence that it is curative. Surgical treatment has been undertaken for certain tumours situated in accessible sites. Thus curettage of the tumour has been performed and subsequent bone grafting undertaken. In other cases amputation has been performed. If the tumour is situated in the spine, and pressure paraplegia supervenes, laminectomy may be required to relieve pressure on the spinal cord.

Pathological aspects—In 1941, one of us (R. A. W.) reported a necropsy case of solitary plasmocytoma of the second cervical vertebra and reviewed examples of solitary plasmocytoma of bone that had been reported previously. Of these, thirteen were accepted as truly solitary, namely those described by Shaw (1923), Walthard (1924), Zdansky (1927), Martin *et al.* (1928), Rogers (1930), Stewart and Taylor (1932), Harding and Kimball (1932), Rutishauser (1933), Chesterman (1936), Cutler *et al.* (1936, Nos. 17 and 18), Leedham-Green *et al.* (1938) and Willis (1941). It was emphasized that a precocious lesion of generalised myelomatosis might appear clinically to be solitary for some months. A tumour can be accepted as truly solitary, therefore, only if no further tumours appear and careful radiographic examination

of the rest of the skeleton remains negative for at least one year thereafter. Alternatively, adequate necropsy examination may prove the solitary nature of the growth, as in the present case and the one reported earlier. Several reported cases that failed to fulfil these requirements were rejected as not being certainly solitary.

Review of recent reports in the literature—Certain relevant reports have appeared, or have come to our notice since the 1941 review was made. Pasternack and Waugh (1939) reported a solitary plasmocytoma of the upper end of the humerus in a man forty-six years old: the tumour had been present for six years; radiographs of the whole of the rest of the skeleton were negative; there was no Bence-Jones proteosuria; and the patient remained well fifteen months after resection of the growth.

King (1940) described an instructive case in which a large, apparently solitary, plasmocytoma of the femur proved to be the forerunner of generalised myelomatosis four years later. Paul and Pohle (1940), under the title "solitary myeloma of bone," described five cases which however had little claim to that designation: two of them showed multiple lesions six and eight months later; and the total periods of observation of the other three were only nineteen, four and eight months respectively. Kirsch (1941) reported a plasma-cell tumour of the femur in a man aged forty-six years in whom radiographs twelve years later showed a lesion in a rib; although this was not proved to be a plasmocytoma, its existence prevents us from regarding the femoral tumour as solitary. Brehant (1941) reported as a case of "plasmocytome solitaire du tibia" a patient who showed Bence-Jones proteosuria, whose other bones were not examined radiographically, and whose history included no follow-up. Esposito's (1943) diagnosis of "solitary myeloma of the skull" applied to a man aged sixty-nine years who was under observation for only five months.

Gootnick's (1945) Case 1 was a man aged forty-eight years, in whom a plasmocytoma of the ilium was treated by curettage and irradiation; there was no radiographic evidence of generalisation over four years later. Gootnick's Case 2 was a man aged sixty-eight years with a plasmocytoma of the ilium treated by irradiation; the patient died of prostatic carcinoma nearly five years later without evidence of myelomatosis (but there is no mention of radiographic examination of the rest of the skeleton).

Schwartz's (1945) paper is mentioned here only as a warning against confusion of nomenclature that should now be extinct: in his paper, entitled "solitary myeloma of the frontal bone," he cited previously reported cases of plasmocytoma, and then reported a case of giant-cell tumour. Tennent (1945) described a large plasmocytoma of the ilium in a male patient aged fifty years in whom radiographs of the rest of the skeleton showed no other lesion, but who was readmitted two years later with generalised myelomatosis and Bence-Jones proteosuria. Blum's (1947) case of "solitary myeloma" has no claim to that title; the patient died five days after hemipelvectomy for a plasmocytoma of the ischium.

The recent paper by Lumb and Prossor (1948) is a useful review of the subject of plasma-cell tumours, and includes three cases of interest in the present connection. One of these, number 6, showed the first signs of multiple lesions four years after the first symptoms of a large spinal plasmocytoma. Case 7 was a man aged forty-eight years who had a plasmocytoma of the upper end of the femur treated by irradiation, who showed no sign of any other tumours radiographically, and remained well for over two years after the first onset of symptoms. Case 8 (also published by Lumb, 1948) was of special interest in that a large solitary plasmocytoma of the sacrum in a man aged seventy-two years (proved solitary by later necropsy) was accompanied by Bence-Jones proteosuria and protein blockage of the renal tubules, a complication hitherto described only in cases of myelomatosis.

From the foregoing review it will be evident that too many cases of plasmocytoma are still being claimed as "solitary" on unsubstantial grounds. This diagnosis can be accepted only if, after the onset of the supposedly solitary growth, there is a long period of freedom from clinical and radiographic evidence of myelomatosis, or if thorough necropsy proves

that no other tumours are present in the skeleton. The length of time that must elapse before a patient with an apparently solitary growth can be declared free from risk of generalisation is uncertain. While most cases of myelomatosis show their multiple character within a year of the onset of a seemingly solitary tumour, there are occasional cases (such as those of King, Tennent, Lumb and Prossor, Case 6, and possibly that of Kirsch) in which evidence of myelomatosis does not appear for several years. In spite of these exceptional cases, there is no doubt from available records that truly solitary plasmocytoma of bone is an entity distinct from myelomatosis, and that it can be cured by adequate local treatment. To those cases accepted as solitary by Willis in 1941, may be added those of Pasternack and Waugh (1939), Gootnick (1945, Case 1), Lumb and Prossor (1948, Cases 7 and 8), and the present case. This would bring the total number of accepted cases up to eighteen. Of these, five had vertebral tumours (Walthard, Cutler *et al.*, Willis, Lumb, Raven and Willis). It is noteworthy that fifteen of the total eighteen cases, including all those with spinal tumours, were men.

TABLE I

SOLITARY PLASMOCYTOMA OF BONE—CASES REPORTED IN THE LITERATURE

Author	Sex	Age	Site of Tumour
Shaw	M	29	Humerus (shaft)
Walthard	M	55	Spine (C.7 and T.1)
Zdansky	F	68	Femur (upper third)
Martin *et al.*	F	56	Femur (upper third)
Rogers	M	34	Femur (shaft)
Stewart and Taylor	M	34	Humerus (upper third)
Harding and Kimball	M	60	Femur (upper third)
Rutishauser	F	62	Femur (upper third)
Chesterman	M	39	Tibia (shaft)
Cutler *et al.* (Case 17)	M	58	Pelvis
Cutler *et al.* (Case 18)	M	52	Spine (L.2)
Leedham-Green *et al.*	M	56	Pelvis
Willis	M	45	Spine (C.2)
Pasternack and Waugh	M	46	Humerus (upper third)
Kirsch	M	46	Femur
Gootnick (Case 1)	M	48	Pelvis
Lumb and Prossor (Case 7)	M	48	Femur (upper third)
Lumb and Prossor (Case 8)	M	72	Sacrum
Raven and Willis	M	56	Spine (T.6 and 7)

It should be added that in cases such as the one now reported, in which after a brief history careful necropsy reveals only one tumour, there is no certainty that, had the patient survived longer, the tumour would have remained solitary. Only one tumour is present now, but multiple lesions might have developed subsequently, since myelomatosis is a multifocal systemic disease. In such cases, then, we cannot know whether the tumour is merely the precocious forerunner of the systemic disease or a true instance of the distinct entity solitary plasmocytoma. The second alternative is perhaps the more probable.

SUMMARY

1. A case of solitary plasmocytoma of the thoracic part of the spine, verified by necropsy, is described.

2. A brief review is given of eighteen acceptable cases of solitary plasmocytoma of bone.

3. Of the eighteen patients, fifteen were men; the five spinal tumours were all in men.

4. Diagnosis requires: *a*) biopsy identification of plasmocytoma; *b*) exclusion of the possibility of generalised myelomatosis by complete radiography of the skeleton, repeated if necessary at intervals during the ensuing two or three years or longer.

5. A tumour of brief duration, proved to be solitary by careful necropsy, cannot be placed with certainty in the group of truly solitary plasmocytomas; it might have been a precocious first lesion of myelomatosis.

REFERENCES

BLUM, S. D. (1947): American Journal of Roentgenology, **57,** 239.
BREHANT, M. (1941): Journal de Chirurgie, **58,** 98.
CHESTERMAN, J. T. (1936): British Journal of Surgery, **23,** 727.
CULTER, M., BUSCHKE, F., and CANTRIL, S. T. (1936): Surgery, Gynecology and Obstetrics, **62,** 918.
ESPOSITO, J. J. (1943): Radiology, **40,** 195.
GOOTNICK, L. T. (1945): Radiology, **45,** 385.
HARDING, W. G., and KIMBALL, T. S. (1932): American Journal of Cancer, **16,** 1184.
KING, B. B. (1940): Journal of the American Medical Association, **115,** 36.
KIRSCH, I. E. (1941): Medical Bulletin of the Veterans' Administration, **18,** 96.
LEEDHAM-GREEN, J. C., BROMLEY, J. F., and RABAN, J. (1938): British Journal of Surgery, **26,** 90.
LUMB, G. (1948): British Journal of Surgery, **36,** 16.
LUMB, G., and PROSSOR, T. M. (1948): Journal of Bone and Joint Surgery, **39-B,** 124.
MARTIN, J. F., DECHAUME, J., and LEVRAT, M. (1928): Bulletin de l'Association francais pour l'étude du Cancer, **17,** 537.
PASTERNACK, J. G., and WAUGH, R. L. (1939): Annals of Surgery, **110,** 427.
PAUL, L. W., and POHLE, E. A. (1940): Radiology, **35,** 651.
ROGERS, H. (1930): British Journal of Surgery, **17,** 518.
RUTISHAUSER, E. (1933): Zentralblatt für allgemeine Pathologie und pathologische Anatomie, **58,** 355.
SCHWARTZ, C. W. (1945): American Journal of Roentgenology, **53,** 573.
SHAW, A. F. B. (1923): Journal of Pathology and Bacteriology, **26,** 125.
STEWART, M. J., and TAYLOR, A. L. (1932): Journal of Pathology and Bacteriology, **35,** 541.
TENNENT, W. (1945): British Journal of Surgery, **32,** 471.
WALTHARD, B. (1924): Schweizerische medizinische Wochenschrift, **54,** 285. (Abstract in Zentralblatt für allgemeine Pathologie und pathologische Anatomie, 1924–25, **35,** 340.)
WILLIS, R. A. (1941): Journal of Pathology and Bacteriology, **53,** 77.
ZDANSKY, E. (1927): Fortschritte der Rontgenstrahlen, **36,** 368.

We are indebted to Miss Irving-Gass for photographs of the specimen and to Mr Cowles for the photomicrograph.

FRACTURES OF THE DORSO-LUMBAR SPINE

E. A. NICOLL, MANSFIELD, ENGLAND

From the Orthopaedic and Accident Service of the Mansfield General Hospital

This paper is based on a study of 166 fractures or fracture-dislocations of the dorso-lumbar spine occurring in 152 miners during the six-year period ended in 1945. One of the objects of the analysis was to investigate the relationship between anatomical and functional results—in other words to examine the validity of the assumption that a good anatomical result is indispensable to a good functional result. This assumption was the basis of the teaching of Böhler (1935) which influenced the practice of most continental clinics; it was reiterated in recent American writings by Key and Conwell (1946); and it has been supported in Great Britain by so great an authority as Watson-Jones (1943) in the words: "perfect recovery is possible only if perfect reduction is insisted upon; even slight degrees of wedging of the vertebrae may cause persistent aching pain. . . ." Nevertheless an assumption it remains, and an assumption that has the most vital repercussions on treatment. The fact that it is held so widely and authoritatively is yet another reason for submitting it to careful and critical examination.

The assessment of deformity is a matter of precision, involving measurable criteria. The assessment of function, however, is based on criteria that are subject to individual judgment rather than precise measurement. Because of the importance of the hypothesis to be examined it seemed necessary to impose an exacting standard for the estimation of function, and certain safeguards were therefore observed : 1) no case was assessed in less than two years, and the average follow-up was five years—long enough to include both late improvement and late complications; 2) the measurement of such physical properties as mobility and muscle power is straightforward but the assessment of pain, which is the most important limiting factor in function, is much more difficult. However, the fact that all these patients were miners provided a satisfactory solution, namely, their ability to withstand the conditions of stress that arise in working at the coal-face in cramped positions. No patient was classified as having gained a perfect functional result unless he could work under these conditions without discomfort and had, in fact, done so for a number of years.

MECHANISM OF INJURY

All the injuries were due to direct violence, usually severe enough to cause contusion and tenderness of the erector spinae muscles, a factor that is of significance in relation to treatment and residual pain. The usual mechanism was hyperflexion which is always associated with a forward shearing stress. The patient was "buried" by a fall of roof or coal with his head between his knees. If the main force falls on the shoulders, the dorso-lumbar region receives the greatest strain; but if the force is applied lower down, the lumbar or lumbo-sacral regions are involved. If the knees are extended at the time, the pelvis is fixed by the tight hamstring muscles and there is much greater stress at the lumbo-sacral region. This was noted in two cases of traumatic spondylolisthesis at this level. In some cases, forward flexion was combined with twisting to one side and this was associated particularly with fractures of the laminae and with the variety of fracture to be described later as "lateral wedge fracture." There were no examples of extension fracture which must be very rare indeed in the dorso-lumbar region, though not so rare in the neck.

Level of injury—Table I shows the distribution of fractures at different levels. Cervical injuries and isolated fractures of the sacrum were not included.

The distribution does not differ materially from that which has been recorded by other writers. The important point to note is that 66 per cent. of the injuries were confined to three vertebrae—the twelfth dorsal, first lumbar and second lumbar.

TABLE I
LEVEL OF INJURY IN 166 FRACTURES OCCURRING IN 152 PATIENTS

Level of fracture	Number of cases	Percentage of total	
Dorsal 10 and above	7	4·2	
Dorsal 11	12	7·2	
Dorsal 12	34	20·5	66·4
Lumbar 1	49	29·6	
Lumbar 2	27	16·3	
Lumbar 3	17	10·2	
Lumbar 4	12	7·2	
Lumbar 5	8	4·8	
Total	166	100·0	

Number of cases with multiple fractures=14 (9%)

Type of fracture—The fractures were classified on an anatomical basis into four main types: anterior wedge fracture, lateral wedge fracture, fracture-dislocation, and isolated fracture of the neural arch (Table II). This differs from the classification adopted by Watson-Jones (1943) who did not differentiate lateral wedge fractures or isolated fractures of the neural arch, and believed that comminuted wedge fractures were relatively common (15 per cent.).

TABLE II
TYPES OF FRACTURE

	Number of cases	Percentage
Anterior wedge fracture	88	58
Lateral wedge fracture	21	14
Fracture-dislocation	29	19
Isolated fractures of the neural arch	14	9
Total	152	100

Anterior wedge fracture—The extent of wedging varies, but in half the cases it is minimal. It cannot, in fact, be severe without involving the posterior interspinous ligament since the fulcrum of movement is at the nucleus pulposus of the intervertebral disc (Figs. 10 and 11). If this fulcrum remains intact, and wedging is severe, rupture of the ligament with subluxation of the posterior intervertebral joints is inevitable. This can often be demonstrated in good radiographs, or it can be inferred from the undue separation of spinous processes. Many so-called wedge fractures with severe wedging are, in fact, fracture-subluxations in which the inferior facets of one vertebra are displaced upwards or even " perched " on the superior facets of the other (Fig. 15). The importance of recognising this lies in the fact that it is an unstable position which is easily converted into a fracture-dislocation with forward displacement of the vertebral body and possible damage to the cord; whereas cases with minimal or moderate wedging and an intact posterior interspinous ligament are quite free from this danger.

There is a special type of anterior wedge fracture in which the whole of the vertebral body is more evenly compressed. This " concertina " type of compression occurs in older people and there is little or no angulatory deformity. As a rule it is associated with

degenerative changes in the nucleus which fails to act as a fulcrum. The interspinous ligament remains intact and vertical compression occurs.

Wedge fracture with comminution—These cases are stated by Watson-Jones to constitute 15 per cent. of all spinal fractures and to be associated especially with the type of direct violence that occurs in mining accidents. Since this was the mechanism responsible for *all* cases in the present series it would have been reasonable to expect a higher incidence, even up to 30 per cent.; but in fact only eight cases were seen in simple wedge fractures (Figs. 1 and 2). Comminution is much more often seen in fracture-dislocations. Its importance in association with wedge fractures lies in the fact that it has been put forward as an explanation of late redisplacement which will be discussed later.

FIG. 1

FIG. 2

Vertical compression fracture showing comminution of the vertebral body and slight retropulsion. There is also an anterior wedge fracture of L.1 without comminution (Fig. 1). The compression was reduced and after six months' immobilisation in plaster the comminuted body healed well. There was no collapse but the disc spaces above and below are narrowed, and spontaneous fusion is occurring (Fig. 2). This is often the case when there is comminution.

Lateral wedge fracture—This is a type of vertebral fracture that has not been described in previous classifications. It constitutes 14 per cent. of the present series and has certain anatomical and clinical characteristics that appear to justify its differentiation. It is a flexion-rotation injury, the usual history being that the patient's head is forced forwards and to one side. The unilateral wedging, the associated fractures of transverse processes on the convex side, and the damage to the posterior intervertebral joint on the concave side, should be noted (Figs. 3 and 4).

Certain clinical features are specially associated with this fracture. 1) The prognosis is not good; in this series only 21 per cent. gained complete functional recovery as against 40 per cent. in anterior wedge fractures. 2) Residual pain, if present, is commonly at the site of fracture (93 per cent.) whereas in anterior wedge fractures pain is usually at the fifth lumbar level regardless of the site of fracture (72 per cent.). The pain in lateral wedge fractures is related either to the displaced intervertebral joint which may also involve the nerve root, or to tearing of the ilio-psoas muscle as indicated by associated fractures of the transverse processes which usually show wide separation, indicating severe damage to the soft tissues. Retroperitoneal haemorrhage is not uncommon and may present all the signs and symptoms of an acute abdominal catastrophe. Two patients in this series with lateral wedge fractures

were submitted to laparotomy, with negative findings (Fig. 3), before the spinal condition was diagnosed. 3) These fractures are difficult to reduce. In fact, reduction was achieved only three times in twenty-one patients; in two of the three there was redisplacement, and the only good anatomical result was secured by grafting. 4) Damage to the cord may occur in lateral wedge fractures whereas there was no such damage in any anterior wedge fracture in this series. There were two paraplegias in twenty-one cases of lateral wedge fracture and both failed to recover. The probable explanation is that the cord and cauda equina are tethered laterally by the nerve roots and dentate ligament, so that lateral flexion is more liable to tear the cord than simple forward flexion. This is supported by the fact that in the two cases mentioned above, paralysis was greater on the side opposite the wedging.

FIG. 3

Lateral wedge fracture with retroperitoneal haemorrhage simulating acute abdomen. If this radiograph had been taken twenty-four hours earlier a fruitless laparotomy might have been avoided.

FIG. 4

Lateral wedge fracture in which there was spontaneous bone fusion. There was considerable deformity but nevertheless the functional result was perfect.

Fracture-dislocation—The essential difference between a fracture-dislocation and a simple wedge fracture lies in the fact that in fracture-dislocation there is rupture of the posterior interspinous ligament. Several degrees of displacement may be distinguished such as simple upward subluxation of the facets, " perching " of the facets, forward dislocation with fracture of the facets or neural arch, and forward dislocation with locking of the facets. All these can be reduced by manipulation except the last, in which manipulation is dangerous. It is important, therefore, to distinguish between them before attempting manipulation and this calls for radiographs of the very best quality. A test reduction may be carried out by turning the patient carefully into the prone position and repeating the lateral view. If there is no reduction in the amount of forward displacement it may be inferred that the facets are locked. Damage to the cord or cauda equina is very frequent in fracture-dislocations (62 per cent. in the present series). There were twenty cases of paraplegia, eighteen associated with fracture-dislocations, and two with lateral wedge fractures.

FIG. 5

Traumatic spondylolisthesis. Closed reduction failed and the patient refused operation. Plaster bed six months. After two years there was spontaneous arrest but the patient still had referred pain in both lower limbs.

FIG. 6

Fracture of the lamina of the second lumbar vertebra. After three years, although there is only fibrous union, forward sliding is minimal. There is no disability and the patient works at the coal-face.

FIG. 7

FIG. 8

Fracture of lamina of third lumbar vertebra. This patient was not seen until six weeks after injury and during that time he had been walking about. Note that there has been no forward sliding (Fig. 7). Three years later there is still no forward sliding and union has occurred (Fig. 8). The patient is now working at the coal-face.

Neural arch fractures—These are rotation injuries, as shown by the fact that half of them are associated with fractures of the transverse processes. They are often described wrongly as fracture of the pedicles—an extremely rare injury which probably never occurs except as part of a severe fracture-dislocation. The site of fracture is at the interarticular part of the lamina so that when there is displacement, as in the bilateral variety, the superior facet slides forwards and the inferior facet remains behind, as in the classical type of spondylolisthesis (Fig. 5).

In bilateral laminar fractures the level of injury is important. If it occurs above the fourth lumbar vertebra the tendency to slide forwards is minimal (Fig. 6). The patient illustrated in Figure 7 was not seen until six weeks after injury, during which time he had been walking about. He was then treated by simple recumbency after which union occurred readily with no further displacement.

At the level of the fourth or fifth lumbar vertebrae the story is quite different, for it is here that body-weight exerts a forward shearing stress. If a bilateral laminar fracture is overlooked at this level (and it is quite easy to do this) forward sliding is bound to result, with consequent spondylolisthesis. The most perfect X-ray films are needed and suspicion should always be aroused if there is a fractured transverse process at this level.

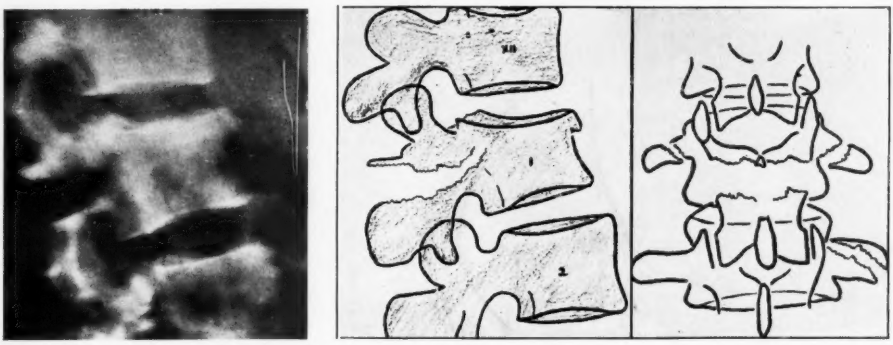


FIG. 9

Chance's fracture, through the spinous process, laminae and vertebral body.

A special type of laminar fracture has been described recently by G. Q. Chance (1948). In this, the fracture line is horizontal and is continued anteriorly into the body and posteriorly into the spinous process (Fig. 9). There may be slight wedging but since the fracture line passes through bone all the way, and there is no subluxation of the facets, or rupture of the posterior ligament, reduction is followed by bony union which is stable. As Chance points out, failure to recognise these cases may lead to unnecessary grafting operations.

REDISPLACEMENT AFTER REDUCTION

Redisplacement after reduction and prolonged immobilisation is more common than is generally supposed. Stanger (1947), reviewing a series of fracture-dislocations of the spine several years after injury, found that deformity had recurred in a high proportion of cases. The same is true of simple wedge fractures and, in the present series, it was the exception rather than the rule for a good anatomical result to be maintained in any case in which initially there had been more than minimal deformity. In the earlier years of the period covered by this review great pains were taken to maintain complete reduction in plaster, sometimes for six months, only to see deformity recur, very often within a month of removing the plaster. The questions that arise are whether the factors responsible for such

redisplacement can be recognised at the outset, and whether a perfect anatomical result is in fact an indispensable objective in treatment.

Mechanism of flexion-extension movement in the normal spine—It is necessary first to consider the mechanism of flexion and extension in a normal spine. The fulcrum of this movement is not at the interarticular joints but at the nucleus pulposus of the intervertebral disc. (Calvé and Galland 1930.) Normally, the posterior part of the disc space actually narrows in hyperextension, proving that this point is behind and not in front of the fulcrum (Fig. 11). If the fulcrum remains intact, any degree of hyperflexion capable of producing even minimal wedging of the vertebral body exerts great leverage on the interspinous ligament. If hyperflexion proceeds still further, rupture of the ligament is inevitable; there may be an avulsion fracture of the spinous process, and a varying degree of subluxation of the facets, depending upon the degree of separation of the spinous processes. This is the sequence of events if the fulcrum remains intact; but in many cases the interspinous ligament is the stronger of the two and the disc itself is crushed.

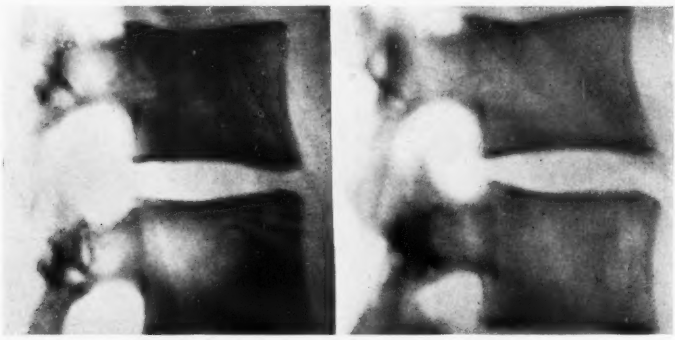


FIG. 10 FIG. 11

The normal mechanism of spinal movement. Fig. 10 shows the intervertebral joint in full flexion and Fig. 11 in full extension. The fulcrum is at the nucleus pulposus.

Thus, four structures are involved—the vertebral body, the disc, the intervertebral joints and the interspinous ligament. Each of these should be considered separately whenever the radiographs of an injured spine are examined. Furthermore, the erector spinae muscles may be overstretched or partly ruptured.

Factors concerned in redisplacement after the reduction of vertebral fractures—The three most important factors concerned in redisplacement can now be considered. They are: comminution of the vertebral body; crushing of the intervertebral disc; and rupture of the interspinous ligament.

Comminution of the vertebral body—Comminution is comparatively rare in simple wedge fractures. When it occurs, consolidation is delayed, as pointed out by Watson-Jones (1943). However, the vertebral body has a good blood supply (evidence of avascular necrosis was observed only twice in the present series) and in about half the patients consolidation of bone in the fully reduced position can be achieved; but the price to be paid is six months in plaster (Fig. 2). Comminution, however, is almost invariably associated with damage to the disc, the interspinous ligament, or both, in which case some collapse is inevitable whatever happens to the vertebral body.

Crushing of the intervertebral disc—This is much more common than is often supposed. It can be recognised in the original radiograph by narrowing of the disc space (Fig. 12) though, after reduction, the space may appear to be normal (Fig. 13). Sometimes the disc can be seen

FIG. 12

FIG. 13

FIG. 14

Vertebral fracture with almost complete disappearance of the disc space indicating that it has been damaged irretrievably (Fig. 12). The disc has no blood supply and is incapable of repair so that deformity is inevitable. Perfect anatomical reduction was maintained for six months (Fig. 13). Nevertheless recurrence of deformity was inevitable—and the inevitable happened three months after removal of the plaster; the disc space collapsed again (Fig. 14).

FIG. 15

FIG. 16

FIG. 17

Often wrongly diagnosed as a wedge fracture with severe deformity (Fig. 15). It is in fact a fracture-subluxation with perching of the facets and rupture of the interspinous ligament. This is an unstable type and should have been grafted. It was fully reduced and immobilised in plaster (Fig. 16). But deformity will recur however long this position is maintained because the interspinous ligament is ruptured. Two years later (Fig. 17) deformity has recurred.

to have prolapsed into the vertebral body and to have remained there after reduction. Lastly, damage to the intervertebral disc may be recognisable only many months later by thinning of the disc space or spontaneous anterior fusion. When the disc is damaged, redisplacement is inevitable, for the disc has no blood supply and it is incapable of repair. Some residual deformity must therefore be accepted and it is not only futile to immobilise damaged soft tissues in an extreme position for long periods in the hope of achieving what is, in fact, impossible, but it is actually harmful to do so. Occasionally, crushing of the disc into the body of the vertebra may cause retropulsion of the posterior part of the vertebral body into the spinal canal (Fig. 1).

Rupture of the interspinous ligament—This ligament may be overstretched, partly torn, or completely ruptured, with or without avulsion-fracture of the spinous process. The significance of these findings is exactly the same as in the case of injuries of the medial collateral ligament of the knee joint. Overstretching and incomplete rupture are compatible with sound healing and restoration of stability; but a completely ruptured ligament seldom heals unless it is sutured. In the case of the spine, it is easier to fuse the spinous processes than to suture the ligament. The degree of damage can be estimated by the amount of separation of the spinous processes in lateral radiographs. In the case illustrated in Figure 15 complete reduction was maintained for six months but nevertheless subluxation recurred within one month of removing the plaster, though not to the original extent.

ANALYSIS OF END-RESULT DISABILITY

1) **Pain**—In this series all the patients were miners and their capacity for work, involving considerable physical stresses and the adoption of cramped positions, was a searching test of function. The miners of Great Britain have special rehabilitation centres, situated in every coalfield, staffed by experienced orthopaedic surgeons and providing a high standard of medical and social service. Spinal injuries are thirty-five times more common in miners than in the rest of the community and as consulting surgeon to this service I have had the opportunity of observing the work of all my colleagues throughout the mining areas.

It has been a universal experience that even the most exacting and prolonged tests in a rehabilitation centre are not equivalent to the functional test of work at the coal-face. Many patients discharged from a rehabilitation centre in the highest grade of recovery fail to stand up to the demands of full work. The reason is always the same—pain returns under the particular conditions of stress and posture that are involved in actual work at the coal-face. Other factors such as mobility, muscle power and muscle endurance are important, but pain under stress is the crucial factor, and as an end-result symptom it calls for detailed analysis. It should perhaps be repeated that no cure was accepted as complete unless the patient had done full work for two years without disability—so that the conclusions may be accepted with some assurance.

TABLE III

ANALYSIS OF 89 CASES WITH RESIDUAL PAIN

Site of pain in relation to type of fracture

Type of fracture	Number of cases	Pain at site of fracture	Low back pain
Anterior wedge fracture .	58	28%	72%
Lateral wedge fracture . .	15	93%	7%
Fracture dislocation . .	7	43%	57%
Fractures of the neural arch .	9	33%	67%
Total . . .	89	40%	60%

Pain is of two types: 1) at the site of fracture; 2) in the low back, independent of the site of fracture. Pain at the site of fracture nearly always occurred in patients with lateral wedge fractures. Of patients who sustained such fractures and had residual pain the localisation was to the site of fracture in 93 per cent. (usually towards the side of wedging), whereas in patients with residual pain after anterior wedge fractures it was localised to the site of fracture in only 28 per cent.

In patients with residual pain after anterior wedge fractures, low back pain is the rule. It occurred independently of the site of fracture in 72 per cent. of these cases. The characteristic features were that it was eased by complete rest, brought on by prolonged sitting or standing, eased by moderate exercise, and brought on again by severe exercise or prolonged forward

stooping. These findings suggest a soft tissue pathology rather than a bone or joint pathology. They are, in fact, identical with those found in cases of low back pain resulting from sprains and contusions and it is probable that they are due to fibrosis and adhesions occurring in damaged muscles and ligaments immobilised in their shortened position. As evidence of this, it was much commoner in patients with anterior wedge fractures treated by hyper-extension plasters (80 per cent.) than in those treated by other methods (37 per cent.). Every surgeon who has operated on these cases in the early stages is familiar with the extensive haemorrhage that is to be found in the erector spinae muscles. This extends to the pelvic attachment, even in high lumbar fractures, because haemorrhage in muscle always tends to gravitate to the most dependent part. It is understandable that pain due to subsequent fibrosis should be felt so commonly at the lower attachment of the muscle.

2) **Mobility**—Reference must be made to loss of mobility as an end-result symptom if only because it has been overstressed in the past. Localised stiffness in the lumbo-dorsal spine causes no appreciable disability, and the test of making the patient bend forwards and put his hands flat on the floor, which is illustrated in many text-books as proof of spinal mobility, is quite fallacious. The radiographs reproduced in Figure 19 are of the patient shown in Figure 18; the greater part of the movement is obviously occurring at the hip joints. Because

FIG. 18

The owner of the spine whose radiographs are depicted in Fig. 19.

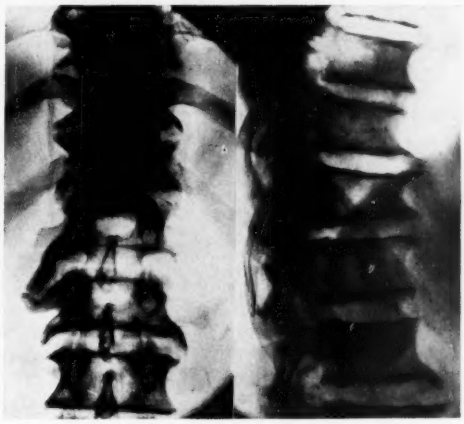


FIG. 19

Ability to touch the toes does not mean that the lumbo-dorsal spine is mobile.

of their occupational conditions most miners retain the long hamstring muscles characteristic of infancy and can perform this test even with a " poker " back. In short, localised fusion of damaged vertebrae, whether the cause be disease or trauma, is the best possible thing that could happen and it causes no significant limitation of movement. If there is gross restriction of movement it is usually due to muscle spasm resulting from pain, and abolition of the pain will restore mobility.

3) **Power and endurance**—Whereas mobility is unimportant, muscle power in the erector spinae group is very important. The miner depends on the strength and endurance of these muscles for his livelihood. These two properties of muscle—power and endurance—must not be confused: they are quite distinct; they have a different physiological basis; and they call for a different technique of treatment in rehabilitation (Nicoll 1948). Pain, however, may inhibit the full use of muscles and prevent their full redevelopment. These functions of muscle can be measured fairly accurately but in doing so it is important to know whether deficiency is due to actual loss of muscle power, or simply to painful inhibition restricting

full output. In the miner, these muscles have to be capable of working at a high level of efficiency for eight hours without respite under conditions that are particularly trying.

RELATIONSHIP BETWEEN DEFORMITY AND DISABILITY

We may now consider the hypothesis, on which present-day orthodox treatment is based, that a good anatomical result is indispensable to a good functional result. The hypothesis will be examined from two aspects: 1) records of individual cases; 2) statistical analysis of the whole series. It is hoped to show that there are no grounds whatever for the above assumption.

Early in 1940, a miner was admitted to hospital suffering from a supraspinatous cuff injury. In the ordinary course of examination it was impossible not to notice that he had a deformed back, and radiographs showed gross deformity (Fig. 20). This fracture-dislocation of the spine had been sustained thirty years previously; it had never been diagnosed or treated and it had united in the position of deformity; the miner has been completely free from symptoms and has worked at the coal-face ever since. This case not only proves that gross deformity is compatible with complete functional recovery, but that there may be no late complications.

FIG. 20

Thirty years ago this man was injured in a pit accident. Treatment was "three months in a feather bed." Every other day his back was rubbed with honey — an ancient remedy mentioned in the Edwin Smith papyrus. It proved so effective that within a year he was at the coal-face and has worked there ever since.

FIG. 21

Vertebral fracture resulting in spontaneous fusion in a position of deformity. The surgeon was mortified at this terrible result but the patient himself was quite pleased with it because he has no pain and no other symptoms and he has been working at the coal-face without interruption for the last five years.

Shortly after this patient was seen, another miner was admitted, suffering from the fracture-dislocation shown in Figure 21. He developed pneumonia soon after admission and his general condition was so serious that, for six weeks, any treatment by which to reduce the deformity was out of the question. At the end of that time reduction was impossible and the deformity shown in the radiograph had to be accepted as the final anatomical result. The spinous process was so prominent that he developed an adventitious bursa over it and could not lie on his back in comfort. This prevented him from carrying out certain exercises, and his treatment at the Rehabilitation Centre had to be interrupted in order to excise the bursa and the underlying bony prominence. He made a perfect functional recovery and has worked ever since as an underground ripper—very heavy work, equivalent in almost every way to work at the coal-face.

FIG. 22

Fracture-dislocation of the spine with severe deformity and incomplete paraplegia. The vertebral body has split vertically and the spinal cord escaped complete section only by slipping between the fragments. Closure of the gap would have been dangerous and over-zealous efforts at reduction might have ended in disaster.

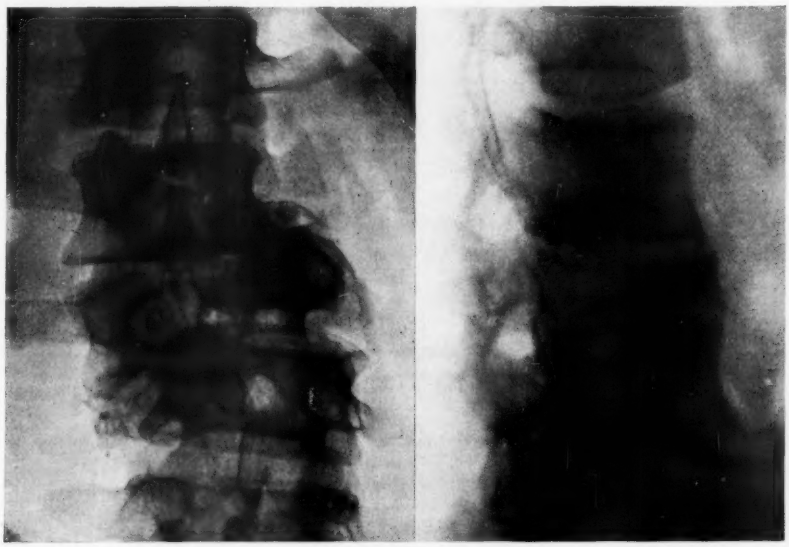


FIG. 23

Same case as shown in Fig. 22, three years later. Spontaneous fusion with marked deformity. The paraplegia recovered and the patient went on to achieve a perfect functional result. He is now working as an underground ripper.

The patient whose radiographs are illustrated in Figure 22 had incomplete paraplegia. There was so much comminution and displacement that complete reduction was impossible. Moreover, enthusiastic attempts at reduction might easily have inflicted further damage on the cord which obviously had escaped total section only by a miracle; it was lying between the two halves of the vertebral body. The patient was simply turned into the prone position and a plaster bed was made in which he was nursed for five months. The final anatomical result is shown in Figure 23. He recovered completely from the paraplegia and resumed full work as an underground ripper. He has no pain in the back and mobility is not restricted significantly.

Lastly, the case illustrated in Figure 24 shows a fracture-dislocation with locking of the facets. There was no damage to the cord. This patient was seen many years ago, before the locked-facet syndrome was well known or the method of dealing with it understood. Naturally, attempts at reduction failed. This man also made a perfect functional recovery and went back to full work at the coal face. He continued to do this for four years without symptoms until a further accident necessitated arthrodesis of the ankle joint.

FIG. 24

Fracture-dislocation of the lumbar spine with locked facets in which stabilisation was achieved spontaneously in the unreduced position. The patient gained a perfect functional result. This radiograph was taken five years after injury.

FIG. 25

Anterior wedge fracture with minimal deformity and no damage to the disc or rupture of interspinous ligaments. Orthodoxy would demand three months in plaster. Fortunately the fracture was overlooked and the patient was back at the coal-face within three weeks.

These examples, which could be multiplied, show that marked deformity is compatible with perfect function. In fact, there were twenty-four patients in the present series who achieved perfect function despite residual deformity. However, any attempt to refute a general hypothesis by quoting individual examples is unscientific. It is always possible that such examples may represent the exception rather than the rule, in which case all that has been proved is that the rule has exceptions. A more scientific method is to examine a series of consecutive and comparable cases. The results in fifty patients in whom recovery, as judged by the very high standard already noted, was classified as complete are analysed in Table IV.

TABLE IV

ANALYSIS OF THE INCIDENCE OF RESIDUAL DEFORMITY IN 50 PATIENTS WITH PERFECT FUNCTIONAL RESULTS

	Good anatomical result	Residual deformity
Anterior wedge fracture . .	20	13
Lateral wedge fracture . .	1	3
Fracture-dislocation . . .	1	8
Fractures of neural arch . .	4	—
Total . . .	26 (52%)	24 (48%)

From Table IV it is clear that the anatomical result is not a significant factor. This is also borne out when the group of anterior wedge fractures is analysed separately. Of eighty-eight patients in this group, fifty-four showed good anatomical results; of these, twenty (37 per cent.) gained perfect functional results. Thirty-four patients had residual deformity and, of these, thirteen (38 per cent.) gained perfect functional results. Table V is an analysis of end-results in relation to treatment, and here a significant factor may be noted.

TABLE V

ANALYSIS OF TREATMENT IN RELATION TO END-RESULTS IN 152 PATIENTS WITH FRACTURES AND FRACTURE-DISLOCATIONS OF THE DORSO-LUMBAR SPINE
(excluding seventeen patients who died or had irrecoverable paraplegia)

End-result (working capacity)	Fixation in plaster	" Functional " treatment
Coal-face	23 (27%)	27 (55%)
Light work underground . .	9 (10%)	13 (27%)
Light work on surface . .	54 (63%)	9 (18%)
Total . . .	86 (100%)	49 (100%)

Of all patients with dorso-lumbar fractures treated in plaster, 27 per cent. were subsequently classified as having gained perfect functional results, whereas, of patients treated by what may be called " functional " methods, 55 per cent. achieved a similarly good end-result. However, analysis of this kind is not really scientific because it might so happen that cases selected for functional treatment had a type of fracture with an inherently better prognosis. The same analysis was therefore made in relation to individual types of fracture (Table VI).

TABLE VI

ANALYSIS OF TREATMENT IN RELATION TO END-RESULTS IN 88 ANTERIOR WEDGE FRACTURES

End-result (working capacity)	Fixation in plaster	" Functional " treatment
Coal-face	10 (20%)	24 (63%)
Light work underground . .	5 (10%)	8 (21%)
Light work on surface . .	35 (70%)	6 (16%)
Total . . .	50 (100%)	38 (100%)

It is seen that in the group of anterior wedge fractures 20 per cent. of patients treated in plaster were classified as having gained perfect results; whereas 63 per cent. of patients treated by " functional " methods gained perfect results. In the other groups, the number of

patients treated functionally was too small to justify conclusions, but there was some evidence in the case of fracture-dislocations that plaster applied in the physiological position gave better results than hyperextension plasters. Magnus (1931), working in the Ruhr Coalfield, came to similar conclusions. He found that in patients treated without fixation in plaster the average duration of incapacity was seventeen weeks and the disability compensation 45 per cent., this comparing with twenty-eight weeks and 64 per cent. respectively for patients treated in plaster.

It is submitted that these findings lead to four conclusions: 1) a good functional result is *not* dependent on a good anatomical result; 2) consolidation is rapid even in the absence of fixation; 3) the important factor in determining function is stability between the damaged segments and not the position in which it is achieved; 4) prolonged fixation of damaged soft tissues, especially in their shortened position, is in itself a cause of disability.

TREATMENT OF DORSO-LUMBAR FRACTURES

Stable and unstable fractures—When there is no special problem of spinal cord injury the best classification, for purposes of treatment, is into stable and unstable varieties. Stable fractures include anterior and lateral wedge fractures and all laminar fractures above the fourth lumbar level. Unstable fractures include all fracture-subluxations with rupture of the interspinous ligament, all fracture-dislocations, and all laminar fractures at the level of the fourth and fifth lumbar vertebrae. In the stable group there is no danger of the deformity increasing and thereby threatening the cord. They should, therefore, be treated " functionally " and not by immobilisation in plaster. In these patients the degree of deformity is never great, it causes no functional disability, and it can be disregarded from the cosmetic point of view. All such patients treated without plaster fixation were examined radiographically on admission, on discharge, and several years later, and in no instance was there significant increase in the deformity. It must be stressed again, however, that nearly all so-called " simple wedge fractures " in which there is severe wedging are, in fact, fracture-subluxations with rupture of the posterior interspinous ligament and are, therefore, unstable.

In the unstable group, deformity is likely to increase and it may even endanger the cord. The high incidence of paraplegia in fracture-dislocations (62 per cent. in the present series) suggests that the cord is always vulnerable in this type of injury. Unstable injuries should therefore be protected against increasing displacement until consolidation is well advanced.

" Functional " treatment of stable fractures—When functional treatment is adopted no attempt is made either to reduce or immobilise the fracture, but the patient is kept in bed for three or four weeks in order to rest the damaged soft tissues and relieve pain. During this period he carries out only extension exercises and receives heat and massage to the back every day to assist the absorption and dispersal of exudates in the muscles and fascial planes. After four weeks he gets up, starts a scheme of full exercises, and is transferred to a Rehabilitation Centre. At the end of eight weeks he has progressed to the highest exercise grade in the Centre and begins a period of hardening which aims at reproducing the stresses and postural difficulties of underground mining. At the end of twelve weeks he should be fit to start work, but often requires another two months on progressively heavy underground work before returning to the coal-face. Some patients beat even this timetable, whereas orthodoxy would demand that every fracture should be reduced and immobilised in hyperextension for at least four months (Fig. 25).

Treatment of unstable fractures—In this series the best results in unstable fractures were gained by applying a " protective " plaster in the neutral or physiological position. The

associated disc or ligament injury makes some degree of residual deformity inevitable. The vertebral bodies are, therefore, allowed to approximate in the plaster so that anterior fusion can occur. In practice, spontaneous anterior fusion with deformity gives a better functional result than surgical fusion. Of ten fracture-dislocations treated on these lines, seven returned to full work at the coal-face; all had spontaneous anterior fusion with deformity and they represent the best results of any group in the present series. No patient whose fractured spine has been treated by surgical fusion has ever returned to coal-face work. The advantages of spontaneous anterior fusion are that it is both stronger and more localised. Posterior fusion is mechanically less sound because the graft is under tension instead of compression. Moreover, undamaged vertebrae are usually included in surgical fusion so that four instead of two become fixed.

The " double clothes-peg " graft described by Bosworth (1942) has removed some of the above objections, and it is now being used to *supplement* anterior fusion, care being taken not to extend the spine beyond the point at which anterior fusion would be prejudiced by distracting the bodies. It is believed that unless there is firm anterior support any posterior graft will eventually give way. A recent series of fifteen unstable fractures treated by the Bosworth graft is being followed-up and will be studied with the object of determining whether or not the long-term functional results are better than those gained by spontaneous anterior fusion.

Treatment of dorso-lumbar fractures with paraplegia—The primary surgical treatment of traumatic paraplegia is still a matter of controversy. In Great Britain a conservative attitude is generally adopted, whereas in the American continent open reduction and grafting is favoured by Rogers, Munro, Botterill, Cone and others. Even among those who favour early operation there is a difference of view regarding the indications. Some would explore almost every case while others, such as Penfield, would operate only on patients with incomplete paraplegia showing a spinal block. On the other hand, Naffziger shares the views of Jefferson, Guttmann, Böhler and others, that paraplegia which is clinically complete from the time of injury and remains so for forty-eight hours indicates that there is complete section of the cord or at least an irrecoverable lesion. Guttmann contends that in these circumstances, paraplegia having been accepted as a permanent disability, the spinal injury should be ignored and all efforts be concentrated on the prevention of pressure sores while stabilisation of the spine in the displaced position is achieved. Böhler points out that this occurs rapidly and that stability can be gained even sooner than would be the case if reduction and grafting had been carried out. However, the premise on which these views are based is not universally accepted and the surgeon who discounts all possibility of recovery within forty-eight hours of injury without the most incontrovertible proof accepts a heavy responsibility.

Many surgeons believe that recovery may occur even after lesions which remain complete for several days. This writer has seen several examples of late recovery in lesions at the cauda equina level, and at least one in which the cord itself was involved, and if this possibility is accepted the lumen of the neural canal should be restored and the spine stabilised in that position in accordance with the dictum of Rogers of Boston. It is generally agreed, however, that any form of plaster fixation, either by means of a plaster cast or plaster bed, is certain to lead to pressure sores so that the maintenance of the reduced position, whether obtained by manipulative methods or by open operation, is difficult. In the absence of plaster fixation, reduction can be maintained only by some form of graft or internal fixation which is self-stabilising, the patient thereafter being nursed on a Stryker frame. If the spinous processes and neural arches are intact, a self-locking letter-box graft can be used. This was adopted recently in two patients who were given no external support and were turned regularly for

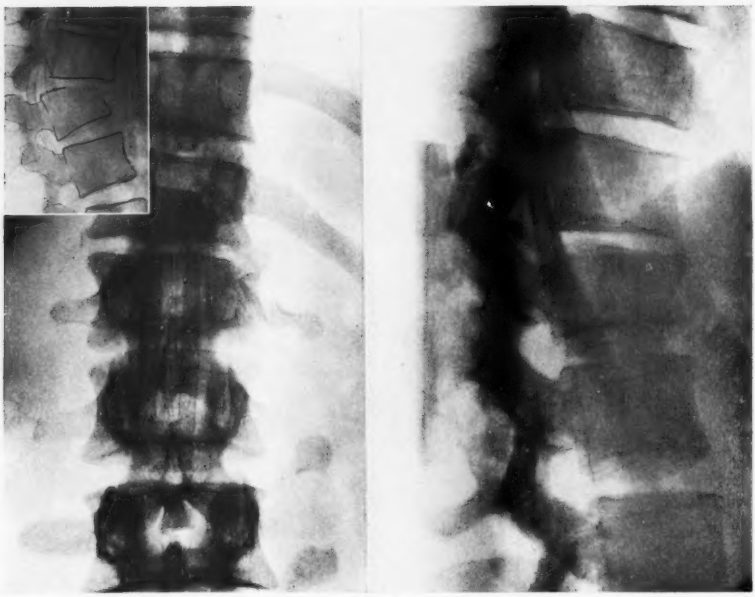


FIG. 26

Fracture-dislocation with paraplegia (inset) treated by self-locking letter-box graft of the spinous processes. No external fixation was used and the patient was turned regularly for nursing care. Ten months later reduction has been maintained and fusion has occurred. Recovery from the paraplegia, in this case, was complete.

FIG. 27

FIG. 28

Traumatic spondylolisthesis (Fig. 27) treated by open reduction and grafting (Fig. 28). This is the *only* way of reducing such cases but it must be done early. Manipulative reduction should not be attempted because it is: *a*) impossible, *b*) dangerous.

nursing care; reduction was maintained until fusion occurred and pressure sores were avoided (Fig. 26). All such methods of self-locking graft depend, however, on the integrity of the posterior neural arches and facets. In three recent cases (one seen by the courtesy of Mr Gordon Irwin of Newcastle) there was such disruption of the neural arches that neither manipulative nor operative reduction could have succeeded in restoring the lumen of the spinal canal and decompression by laminectomy appeared to be justified, especially when there was evidence of spinal block. The primary treatment of traumatic paraplegia is obviously a subject about which there is still much to be learned, and in the present state of our knowledge any dogmatic recommendation is unjustified.

Treatment of isolated fractures of the neural arch—The treatment of fractured laminae depends upon the level of injury and the stage at which the case is first seen. Many of these fractures are overlooked because they demand a very high standard of radiography, especially in the lumbo-sacral region. It has already been stated that above the level of the fourth lumbar vertebra there is no danger of forward sliding beyond minimal limits so that these cases can safely be treated by functional methods. In the present series there were six such cases and four were classified as complete cures. If the lesion is at the fourth or fifth lumbar level, treatment depends upon whether or not there is spondylolisthesis. If not, union will probably occur with four months' immobilisation on a plaster bed; but if forward sliding has already begun, union is unlikely to occur because the conditions resemble those of a gap fracture. Open reduction and fusion is then the best treatment. The method of closed reduction advocated by Watson-Jones (1943) was tried twice but there was no improvement in the position and in one case a temporary foot-drop resulted.

In the present series, there were eight patients with fractures of the laminae at the low lumbar level and of these, six had already begun to slide when first seen. Progressive displacement was prevented in all of them by grafting but none was able to resume strenuous underground work because of low back pain under stress, though all were able to do lighter forms of underground or surface work. The best result was in one whose fracture of the laminae was not displaced but failed to unite after immobilisation in a plaster bed. Operation was then undertaken and fusion occurred in a good anatomical position. From the total of fourteen cases at all levels, there were only five perfect results and all these were good anatomical results. It does seem, therefore, that in this particular type of fracture a good anatomical result is essential. Recently, a case of traumatic spondylolisthesis with considerable immediate displacement was seen and operated on within a few days. It was possible to secure complete reduction and this has been maintained (Figs. 27 and 28). It is now considered that this is the *only* way of obtaining reduction in these cases; but it must be done early.

Treatment of lateral wedge fractures—Lateral wedge fractures present a difficult problem in treatment. Of the twenty-one examples in the present series two were complicated by permanent paraplegia and are, therefore, excluded from the results. Of the remaining nineteen, five were treated functionally with one perfect result. Twelve were treated by immobilisation in plaster, with three perfect results, all of which had residual deformity and one of which showed spontaneous fusion (Fig. 4). Two were treated by immediate surgical fusion; one gained a perfect anatomical result; but neither was able to return to coal-face work because of pain. This experience has been repeated in two other patients who have not yet been observed long enough to qualify for inclusion in the present series. Although the total number of cases is small, it seems justifiable to conclude that since these are stable fractures, since orthodox treatment in plaster does not correct the deformity, and since a good anatomical result secured by grafting does not lead to a perfect result, functional treatment should be the method of choice. Any conclusions based on such a small number of cases, however, must be regarded as tentative, and the final answer to this problem must await the result of further experience.

CONCLUSIONS

Certain types of dorso-lumbar fracture are stable and the deformity will not increase if there is reasonable protection in the early stages. In some of these (*e.g.*, dorsal fractures and lateral wedge fractures) the deformity cannot be corrected in any case, whilst in others it will inevitably recur if it *is* corrected. The final anatomical result can be assessed accurately at the outset and, if this is within limits known from experience to be compatible with perfect function, there is no case for immobilising damaged soft tissues in an extreme position for many months—a procedure which in itself is likely to produce disability.

In unstable fractures, deformity may increase beyond functionally insignificant limits and may jeopardise the cord in so doing. More rigid protection is then necessary, but the aim should be to produce spontaneous anterior fusion in the best anatomical position compatible with that objective, and this is hindered rather than assisted by extreme hyperextension maintained for long periods. Sometimes it may be necessary to reinforce anterior by posterior fusion, but it is doubtful whether posterior fusion alone will ever stabilise a spine that lacks anterior support.

SUMMARY

1. A series of 166 fractures and fracture-dislocations of the dorso-lumbar spine has been reviewed.

2. A new method of classifying these injuries is suggested.

3. A type of fracture with lateral wedging, previously unidentified, which has certain distinctive clinical and anatomical features is described.

4. The factors responsible for redisplacement are discussed and it is considered that in most cases this is predictable from the outset.

5. At the present time orthodox treatment is based on the assumption that a perfect anatomical result is indispensable to a perfect functional result. Analysis of the results in the series now reported shows that there are no grounds for this assumption.

6. Treatment is discussed in the light of the foregoing conclusions. This is based on a division of cases into stable and unstable types, the recognition of which is of crucial importance.

REFERENCES

BÖHLER, L. (1935): The Treatment of Fractures. Fourth English edition. Bristol: John Wright & Sons.
BOSWORTH, D. M. (1942): Surgery, Gynecology and Obstetrics, **75,** 593.
BOTTERILL, H. (1946): Proceedings of the National Research Council of Canada.
CALVÉ, J., and GALLAND, M. (1930): Revue d'orthopédie 3e, série **17,** 5.
CHANCE, G. Q. (1948): British Journal of Radiology, NS **21,** 452.
CONE (1946): Proceedings of the National Research Council of Canada.
GUTTMANN, L. (1948): Personal communication.
JEFFERSON, G. (1936): British Medical Journal, **2,** 1125.
JEFFERSON, G. (1949): Personal communication.
KEY, J. A., and CONWELL, H. E. (1946): The Management of Fractures, Dislocations and Sprains. Fourth edition. St Louis, Missouri: C. V. Mosby Co.
MAGNUS (1931): Hefte zur Unfallheilkunde, **8,** 31, 59.
MUNRO, D. (1948): American Journal of Surgery, **75,** 3.
NICOLL, E. A. (1948): Journal of Bone and Joint Surgery, **30 B,** 392.
PENFIELD, W. (1946): Proceedings of the National Research Council of Canada.
SCHMIEDEN (1931): Hefte zur Unfallheilkunde, **9,** 4, 59.
STANGER, J. K. (1947): Journal of Bone and Joint Surgery, **29,** 107.
WATSON-JONES, R. (1943): Fractures and Joint Injuries. Third edition, 2 Vols. Edinburgh: E. & S. Livingstone, Ltd.

ATLANTO-AXIAL FRACTURE-DISLOCATION

KURT COLSEN, JOHANNESBURG, SOUTH AFRICA

From the Department of Surgery of the University Witwatersrand, Johannesburg

A young adult Indian male had complained for two years of fleeting pain in many joints with more or less continuous pain in the cervical region but no limitation of movement. He had been treated for " rheumatism " by several doctors. In January 1947 manipulation of the neck was advised and carried out at another hospital under sodium pentothal anaesthesia. On recovering from the anaesthetic the patient complained of excruciating pain in the midline of the neck beneath the occiput, which was quite different from the pain he had suffered before the manipulation. The head was fixed in a position of flexion with rotation to the left and there was a tingling sensation over the ulnar aspects of the forearms and hands, the medial aspects of the thighs, and the abdomen. He was kept in bed. Five days later he left the hospital, consulted this writer, and was admitted to the Department of Surgery of the University Witwatersrand, Johannesburg.

It was observed that he walked cautiously and deliberately. The head was held in acute flexion with slight rotation to the left. There was spasm of the cervical muscles, especially the paravertebral group, and movement was very limited and painful. Neurological examination revealed no objective signs though he complained of paraesthesia in both forearms and hands. Radiographs showed forward dislocation of the atlas on the axis with a fracture of the odontoid process. The atlas was tilted 60 degrees; the posterior ramus lay on the antero-superior surface of the body of the axis and there was lateral displacement to the full width of the atlas. The odontoid process of the axis was fractured at its base and displaced forwards and downwards with the atlas (Figs. 1, 4, 5).

The displacement was reduced on January 27, 1947, under intratracheal anaesthesia. A skull calliper was applied, the patient being placed on the operating table in such a way that the neck was supported on a wooden board, six inches wide, projecting from the end of the table. The cervical spine was extended and a Böhler screw-traction apparatus (of the type designed for fractures of the lower limb) so placed that the patient's shoulders rested against the upright bars of the frame, the cord from the calliper being attached to the hook of the wing screw (Fig. 6). The dislocation was reduced very gradually by tightening the winged screw and manipulating the frame in the desired position of traction. This manoeuvre lasted one and a half hours, during which time four radiographs were taken to check progress of the reduction (Fig. 2). When a satisfactory position had been secured a plaster jacket was applied. The skull calliper was left in position and weight-traction was continued for four weeks. Subsequently the calliper was removed and a new plaster applied. At this time there were no neurological symptoms and the patient was able to walk without discomfort.

Because of the notorious instability of this type of dislocation, cervico-occipital fusion was performed sixteen weeks later. A window was cut in the back of the plaster, and the base of the occiput and upper four cervical vertebrae were exposed through a midline incision. A gutter was made in the occipital bone by removing a strip of the external table, and the spinous processes of the second, third and fourth cervical vertebrae were split to receive a bone graft cut from the iliac crest. The graft was held in position by sutures of tantalum wire.

Three months later, radiographs showed that although there had been some recurrence of forward tilting of the atlas the position was acceptable and there was satisfactory consolidation of the graft. The plaster was discarded and a wool collar of Schanz type was worn for a further six weeks. At this time the head could be moved through about half the normal range and there was no pain on movement. Eighteen months later the patient reported

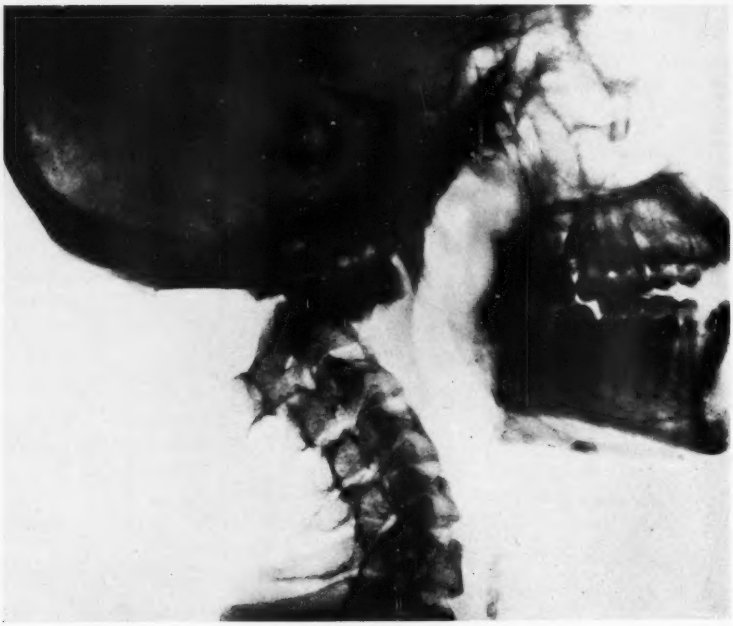


FIG. 1

Fracture-dislocation of the atlanto-axial joint occurring after manipulation for "rheumatism" of the neck. The odontoid process is fractured and displaced forwards with the atlas (see also Figs. 4–5).

FIG. 2

FIG. 3

Reduction was effected by screw-traction combined with hyperextension (Fig. 2). Cervico-occipital fusion was then performed with a bone graft and the condition fourteen weeks after operation is shown in Fig. 3. The graft is well consolidated.

that he had no symptoms and neurological examination showed no abnormal findings. Flexion movement was limited by approximately 15 per cent., extension by 30 per cent., and rotation by 50 per cent. Radiographs showed sound consolidation of the graft from the occiput to the third cervical vertebra.

DISCUSSION

The classical account of " rotatory dislocations of the atlas " by Corner (1907) included a review of twenty cases, eighteen from the literature and two of his own. This paper led to fuller understanding of the mechanism of these dislocations. Corner emphasised the laxity of the atlanto-axial joint which allowed considerable movement without dislocation, and the fact that the muscles of the neck provided considerable protection against injury. If these muscles are relaxed, the very lax atlanto-axial joint is susceptible to injury. Lambotte (quoted by Mixter and Osgood 1910) reported the case of a young woman who sustained

FIG. 4

FIG. 5

Tracings of the original displacement of the atlas and odontoid process.

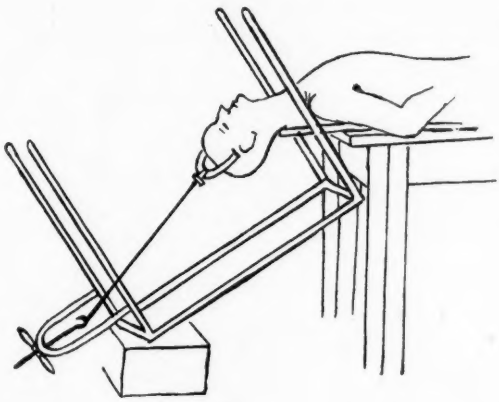


FIG. 6

Reduction by screw-traction was secured by means of the Böhler limb-traction frame.

complete dislocation of the atlas, with fracture of the odontoid due to a sudden movement of the head while sewing. Wilson (1907) described a case of atlanto-axial injury in which it was believed that the displacement was caused by an osteopath who manipulated the patient's neck in the erect position.

Atlanto-axial dislocation may cause cervical cord injury and death, either immediately or after a delay of hours, days, months or even years. Broca (1863) reported a case proved at autopsy; the patient was an old man who died of urinary disorder and during life had carried his head obliquely. Bernstein (1903) reported a fracture-dislocation between the atlas and axis in which evidence of cord damage was delayed until the seventy-first day. Paralysis began in the right upper and right lower limb and spread slowly to involve the limbs of the left side and the bladder, rectum and diaphragm; the patient died after one hundred and one days. At autopsy there was evidence of unilateral forward displacement of the atlas with callus formation at the site of the fractured base of the odontoid process, compressing the cord. Costes (1855) described a similar case. The patient was progressing well until the ninth week when paralysis began; death occured in the eleventh week. Gibson (1885) reported a patient with dislocation between the atlas and axis with fracture of the odontoid process and clinical features similar to those of the case reported in this paper. The head was set

forward with the chin resting rigidly on the sternum, and there was a prominence at the back of the neck below the occiput. There were no abnormal neurological signs. The dislocation was reduced by steady traction. One week later, against advice, the patient sat up and immediately fell back dead. Autopsy showed separation between the axis and atlas with the cord tightly stretched and pulled against the anterior wall of the canal. The odontoid process was broken off at its base and displaced forwards with the atlas. Another case was described by Elliot and Sachs in 1912; their patient died from paralysis after thirty-two years, " a long earthly existence with a broken neck." Watson-Jones (1943) considered that dislocation alone was more serious than fracture-dislocation, " because if the odontoid process is intact the spinal cord is in danger of being crushed against it."

From these references it is evident that atlanto-axial fracture-dislocation is not necessarily fatal and may even occur without cord-symptoms. Nevertheless, in view of the extreme degree of displacement in the case here reported it seems almost miraculous that the patient suffered only minimal and temporary cord-symptoms and that apart from some limitation of movement in the upper cervical spine he is now entirely free from disability.

Treatment—Reduction of the displacement may be secured slowly by continuous weight-traction with a skull calliper, or by a one-stage manoeuvre. The latter method was preferred in this case. Immobilisation may be maintained by plaster but there is danger of recurrence of the deformity. Cone and Turner (1937) described both immediate and late re-displacements, causing delayed nerve-root or spinal-cord symptoms. Re-displacement and death have been reported after prolonged immobilisation in plaster either suddenly, as for example when the patient sneezed, or gradually by increasing recurrence of deformity.

This instability of the reduction of fracture-dislocations can be overcome by cervico-occipital fusion. Several successful cases have been described (Cone and Turner 1937). In the case now reported a graft from the iliac crest was used and the difficulty of maintaining contact of the graft with the occiput was overcome by the use of wire sutures of tantalum wire.

SUMMARY

1. A case of fracture-dislocation of the atlas on the axis is presented in which the neck had been manipulated under anaesthesia for " rheumatism."
2. Despite marked displacement there were no important neurological complications.
3. One-stage reduction by traction was carried out very slowly and with radiographic control. It is believed that this is less distressing to the patient, and safer, than gradual reduction by prolonged traction.
4. Because of the instability of the atlanto-axial joint after reduction surgical fusion of the upper cervical spine to the occipital bone is advisable.
5. In the case now reported recovery was complete.

The author wishes to express his grateful appreciation to Mr P. Keen and Mr R. J. Fleming for their help in treating this patient and to Miss P. Bilborough for the photographs.

REFERENCES

Bernstein, R. (1903): Deutsche Zeitschrift für Chirurgie, **70,** 175.
Broca, P. P. (1863): Bulletin de la Société de Chirurgie, 2e série, **3,** 549.
Colsen, K. (1942): Fractures and Fracture Treatment in Practice. Johannesburg: Witwatersrand University Press.
Cone, W., and Turner, W. G. (1937): Journal of Bone and Joint Surgery, **19,** 584.
Corner, E. M. (1907): Annals of Surgery, **45,** 9.
Costes (1855): Cited in Malgaine, J. F.: Traité des Fractures. Paris: J. B. Baillière, **2,** 329.
Elliott, G. R., and Sachs, E. (1912): Observations on Fracture of the Odontoid Process of the Axis with intermittent pressure paralysis. Annals of Surgery, **56,** 876.
Gibson, W. W. (1885): Lancet, **2,** 429.
Mixter, S. J., Osgood, R. B. (1910): Traumatic Lesions of Atlas and Axis. Annals of Surgery, **51,** 193.
Watson-Jones, R. (1943): Fractures and Joint Injuries. Edinburgh: E. & S. Livingstone, Ltd.
Wilson, H. A. (1907): Annals of Surgery, **45,** 632.

SURGICAL ASPECTS OF THE TREATMENT OF TRAUMATIC PARAPLEGIA

LUDWIG GUTTMANN, OXFORD, ENGLAND

From the Spinal Injuries Centre, Ministry of Pensions' Hospital, Stoke Mandeville, Aylesbury, Buckinghamshire

In the treatment of vertebral injuries with spinal cord involvement the views expressed in recent years by surgeons, some of whom advocate exploratory laminectomy whereas others favour conservative treatment, are no less controversial than they were in the time of Astley Cooper and Charles Bell, one hundred and twenty-five years ago. Even among those who advocate surgical intervention the best time for laminectomy is still a matter of some disagreement, and this applies both to early and late operations.

In assessing the indications for surgical intervention it is essential first to make a clear distinction between traumatic paraplegia due to closed spinal injuries and paraplegia due to compound spinal injuries such as stab wounds and, particularly, gunshot and shell wounds. Moreover, the term " early treatment " must be defined more clearly than has been done in the past. It has been interpreted differently by various authors. Some, when they refer to " early laminectomy," mean exploration within forty-eight hours of injury, whereas others believe that operation after one to two weeks is still " early." A distinction should be made between surgical intervention at the most acute stage, within the first three days, and operation during the next two to three weeks. Surgical intervention within the first three days may be termed " immediate operative treatment " as opposed to " early operative treatment " when the most acute stage has passed.

IMMEDIATE OPERATIVE TREATMENT

Open spinal injuries—In open or compound spinal injuries, regardless as to whether or not they have caused complete or incomplete transverse spinal syndromes, immediate operative treatment is indicated as soon as general shock has been overcome, provided only that there is no associated injury to the lungs or other internal organs. It should usually consist only of debridement. Haematomata and foreign bodies such as bullets, pieces of cloth and bone splinters should be removed whenever possible. The removal of such foreign bodies is indeed vital if there is leakage of cerebrospinal fluid. Dural tears should be closed and penicillin and streptomycin applied locally. If the dura is found to be intact it should not be opened unless there is absolutely clear evidence of a localised subdural haematoma: if there is such a haematoma and if, as in the case of cauda equina lesions, it involves the anterior and posterior roots, these should be disentangled. Such procedure was adopted during the recent war by most British and American neurosurgeons and, as a rule, it proved satisfactory.

Closed spinal injuries with complete paraplegia—In closed spinal injuries due to fractures or fracture-dislocations this writer is in complete agreement with those who advocate conservative treatment and are strongly opposed to laminectomy as an immediate measure. This applies to injuries with complete transverse spinal syndromes at any level, including cauda equina lesions. It may be emphasised that at this stage the Queckenstedt test is of no diagnostic value in differentiating subarachnoid block caused by oedema of the injured spinal cord or pressure from the dislocated bone. Moreover, in complete transverse lesions in which flaccid paraplegia remains unchanged for forty-eight hours there has usually proved to be either complete crushing and destruction of the cord or at least most severe damage which is irreparable.

Importance of instituting rehabilitation immediately—These are the cases in which all efforts should be concentrated on rehabilitation from the very beginning. The main points which have to be considered even in the immediate and early stages are: the prevention of pressure sores; the control of urinary infections; the prevention of contractures of paralysed limbs

due to faulty position, such as keeping the legs constantly adducted, the hips and knees flexed, and the feet and toes in plantar flexion; and the development of the muscles of the trunk and upper limbs on which the patient will have to rely. Everything should be done in these early stages to encourage development of readjustment forces in mind and body to compensate for loss of function in the paralysed parts of the body by increasing the action of the normal parts. Details of the management of patients with traumatic paraplegia have been published elsewhere (Sandifer and Guttmann 1944, Guttmann 1945, 1946, 1947 and 1949).

Dangers of plaster casts and plaster beds—The application of plaster casts is contra-indicated because it leads almost invariably to the development of deep pressure sores. This does not mean that the spine should be ignored completely, as was advocated by Magnus in Germany. From the point of view of rehabilitation it is advisable to bring a badly displaced spine into the best possible position but this can usually be maintained by pillows or blankets without the application of plaster. Plaster beds should be allowed only for transport and be discarded at the earliest possible date. At the beginning of the recent war, plaster beds were recommended for the prevention and healing of pressure sores in patients with traumatic paraplegia, the idea being that pressure was then distributed more evenly. This concept did not prove to be correct; the volume of the paralysed parts does not remain constant because there are changes in the degree of vasodilatation from interruption of the spinal vasomotor centres. In fact, in those patients with paraplegia who lay in plaster beds for months—even when these beds had been constructed by experts—not only did this method of nursing prove to be no better in the prevention and treatment of pressure sores but it actually promoted the development of sores of the most frightful type. In addition, this type of fixation may cause profound contracture of joints, distortion of the pelvis, and atrophy of the back muscles in normal parts of the body which are so vital for physical readjustment and particularly for the later maintenance of the upright position. Moreover, stagnation in the renal system caused by prolonged recumbency and immobilisation may have devastating effects on the bladder and kidneys. It has often taken months and even years of hard work to remedy, or at least to diminish, the damage caused by this form of fixation. The conclusion drawn by the author from his own experience during and after the recent war is that the use of plaster beds, except for the purpose of transport, is contrary to the fundamentals of the rehabilitation of patients with traumatic paraplegia.

Closed spinal injuries with incomplete paraplegia—In closed spinal injuries with incomplete cord or cauda equina lesions this writer also favours conservative treatment in the period immediately after injury; as a rule, operative intervention can be postponed safely. There are, however, extremely rare instances of rapidly increasing epidural haematoma in which operative intervention is indicated at this early stage. An excellent result thus obtained was described by McLean (1935) in a case of a fracture-dislocation of the eleventh dorsal vertebra. Twelve hours after injury the sixteen-year-old patient showed only marked tenderness in the eleventh and twelfth dermatomes with analgesia in the distribution of the first and second lumbar nerves. Sensibility in the saddle area was normal, as were the lower limb tendon reflexes. The symptoms gradually increased and thirty hours after the accident there was almost complete paraplegia with absent reflexes and sensory loss which was more marked in the lumbar regions than in the sacral regions. At operation, forty-eight hours after injury, dislocation of the vertebra was confirmed and partly corrected, and an epidural haematoma that was compressing the cord was evacuated. A posterior plaster shell was moulded to the patient before he was moved from the table. The clinical signs receded within a period of nine weeks. At the thirty-eighth week there was a residual, incomplete Brown-Sequard syndrome.

EARLY OPERATIVE TREATMENT

Open spinal injuries—In compound spinal injuries, particularly those due to gunshot and shell wounds, the main purpose of early operative intervention is the removal of foreign

bodies, especially when there is leakage of cerebrospinal fluid with X-ray evidence of a foreign body within or in the neighbourhood of the spinal canal, and bacteriological evidence of infection of the cerebrospinal fluid. In such a case, removal of the foreign body is vital. A soldier was admitted to this Spinal Centre from the battle-front in Germany on December 11, 1944, fourteen days after being hit in the back by fragments of an 88 mm. shell which burst near him. He had a complete transverse lesion at Th.11, with flaccid paraplegia. There was a wound measuring 4 centimetres by 2 centimetres to the left side of the tenth thoracic vertebra which was discharging cerebrospinal fluid. Bacteriological examination showed infection with clostridia Welchii, B. haemolytic streptococcus and B. coliform bacilli. Radiographs showed a large metallic foreign body in the region of the spinal canal at the level of Th.11 which was fractured. On December 16, I removed a metallic foreign body measuring 4 centimetres by 1·5 centimetres. No attempt was made to close the dura. Post-operative treatment consisted of daily dressings with local penicillin. The wound healed gradually, and the patient, although still paralysed, is now very fit and is gainfully employed as a commercial artist.

Closed spinal injuries—In closed spinal injuries the indications for laminectomy during the early stages are:

1) Incomplete lesions showing progression of the neurological signs. In the writer's opinion, the presence of bone protruding into the spinal canal alone is no indication for surgical intervention in the early stages.

2) Permanent manometric block without evidence of fracture or fracture-dislocation of the spine. In such cases, which are very rare, laminectomy is justified in the early stages irrespective of whether the transverse spinal syndrome is complete or incomplete.

3) Severe and constant irritation of spinal roots caused by displacement of bone fragments or prolapse of intravertebral discs. This, however, is very rare in the early stages. Elsberg (1940), for instance, in twenty years saw only one case of incomplete spinal lesion with root pain sufficiently severe to indicate early surgical interference. In my own series of 370 patients with traumatic paraplegia treated during and after the recent war at Stoke Mandeville I have seen not a single case in which such surgical intervention was justified.

LATE OPERATIVE TREATMENT

Laminectomy in the late stages of traumatic paraplegia has been carried out to serve three main purposes: *a*) restoration of neural function; *b*) treatment of intractable pain; *c*) treatment of violent flexor or extensor spasms.

Restoration of neural function—As a general rule "late laminectomy" does not serve any useful therapeutic purpose in complete transverse lesions at any level. On the contrary, by weakening the stability of the spine and particularly the strength of those muscle groups of the back which are so essential for the upright position, it only delays the rehabilitation of the paraplegic patient to a useful social and industrial wheel-chair life. Moreover, the post-operative shock has a most harmful effect during the first few days on the peripheral vasomotor control in the paralysed parts, thus causing lowered tissue resistance to pressure and greatly increasing the danger of pressure sores. This conception is at variance with the opinion, generally held, that exploratory laminectomy is harmless.

No single patient admitted to the Spinal Injuries Centre at Stoke Mandeville with a complete transverse lesion, due either to closed or open spinal injury, had gained any recovery of neural function of the damaged spinal cord by exploratory laminectomy performed before admission. T. B. Dick (1949) compiled statistics relating to twenty-seven patients subjected to laminectomy more than seven days after injury at the Spinal Injuries Centre, Winwick. Twenty-two had complete lesions before operation and, although only two proved to be anatomically complete, no patient showed evidence of later recovery. Five operations were performed on patients with incomplete lesions: three showed doubtful improvement which probably, as the author states, was not attributable to the operation. Our observations are

in accord with those of McCravey (1945) and Cutler (1945), and it may be noted that in December 1944 the American Surgeon-General directed that late laminectomies in this type of spinal injury should no longer be carried out because it was considered useless in the attempt to restore neural function.

Furthermore, late laminectomy should not be carried out indiscriminately in incomplete transverse lesions. This writer is opposed to the recent recommendation that exploration is indicated even in the presence of neurological improvement if there is radiographic evidence of laminal damage (Haynes 1946). There is no hurry whatsoever, and it is nearly always safe to wait at least until the progress of recovery has ceased.

On the other hand, there is general agreement that laminectomy is indicated in incomplete transverse spinal syndromes when there is evidence of increasing neurological signs. The underlying cause of such clinical progression may be callus formation, or localised chronic pachymeningitis or leptomeningitis (arachnoiditis chronica progressiva cystica adhesiva). Riddoch (1927) and other authors have described cases of post-traumatic chronic meningitis in which the post-operative results were satisfactory. Foerster (1929), though mentioning the satisfactory result of operation in several of his own patients, emphasised that in most patients with chronic post-traumatic arachnoiditis improvement in the spinal symptoms after operation was not very impressive; and he assumed that post-traumatic thrombosis of the spinal cord vessels was often responsible for irreversible lesions of the cord. There are also selected cases of osteomyelitis with local pachymeningitis in which exploration may be indicated. In one of my own cases in this series, in a naval officer, there was an incomplete lesion at the mid-thoracic region and, at operation, a track was found running down to the vertebra associated with localised pachymeningitis and very marked increase of fibrous tissue which was removed from the dura. The result of the operation was satisfactory in so far as there was relief from pain, and arrest of the increasing motor weakness, although the effect on the spasticity was insignificant.

Treatment of intractable pain—Pain is most frequent in cauda equina and distal thoracic cord injuries and is mainly of the causalgic type. It also occurred in patients in this series, with complete or incomplete cervical lesions, who were admitted with serious contractures of the elbow and shoulder joints. The pain may be general or it may be referred to definite parts of the limbs or trunk. It is often agonising and the life of the patient becomes almost unbearable. The treatment usually given to patients with such pain before admission to this Centre had often included long continued injections of morphine and other narcotics and, sometimes, sympathectomy or cordotomy.

The approach of this writer to the problem of pain in paraplegia, based on dissatisfaction with his own previous operative results with posterior rhizotomy and cordotomy, has been quite different from the beginning. The essential principle of treatment is to mobilise, and develop to the highest possible level, readjustment forces in the mind and body of the patient in order to master the various symptoms of his disability, including pain. This is achieved by appropriate psychological measures, healing and prevention of septic conditions, treatment of anaemia caused by sepsis, frequent passive movements and all forms of active physiotherapy including recreations and, above all, pre-vocational training which has proved the best possible form of occupational therapy by which to counteract frustration. The prescription of morphia and other heavy narcotics which obviously impedes the mobilisation and development of these adjustment forces was reduced to a minimum, and in due course was almost completely abandoned. Some years ago Gowlland (1934), when writing of the treatment of paraplegic patients, stated: " I suppose that there is more morphia, atropine and hyoscine used in the Home which I look after than in any other place of the same size in the country." He continued: " One of the snags is that some of these poor fellows who really do suffer and whose pain has been relieved by morphia, are apt to become addicts." In striking contrast to this approach, which still prevails in many centres for the treatment of paraplegia, it can be

stated that the Spinal Centre at Stoke Mandeville Hospital is one of the medical institutions where the *least* amount of morphia and other heavy narcotics is used for the treatment of painful conditions. In some cases pain has not been entirely eliminated, but this has in no way prevented successful rehabilitation. Repeated paravertebral injections of novocain have sometimes been employed and in two patients, where pain was associated with severe abdominal spasm, intrathecal injections of alcohol were successful. It may be concluded that radical surgical procedures such as sympathectomy, posterior rhizotomy, cordotomy, and posterior column tractotomy have very limited application in the treatment of pain in traumatic paraplegia. Surprising as it may seem, the apparently intractable pain is best relieved by general rehabilitation and retraining of the patient.

Treatment of violent flexor or extensor spasms—In the past, severe flexor or extensor spasms were considered to be one of the most devastating complications of spinal cord injury, preventing rehabilitation and making the life of the patient intolerable. During the last five years much research has been undertaken by which to distinguish the various causes underlying the mechanism of this spasm, and great progress has been made in treatment. It can now be concluded that if adequate care and appropriate preventative measures are instituted *at an early date* after injury, exaggerated reflex activity of the paralysed limbs never becomes so severe that ambulation and rehabilitation is prevented. The spasticity can be kept in check either by conservative methods or by simple operations such as lengthening of the tendo Achillis and neurectomy of the obturator nerves. Obturator neurectomy has proved to be an excellent method of restoring the sexual activities of the paraplegic patient by eliminating adductor spasm which is the main obstacle to intercourse. In patients who were admitted at *later dates* after injury, with profound reflex spasm of the paralysed limbs and resulting contractures, and whose previous treatment had consisted mainly of the administration of morphine and other narcotics, intrathecal injection of alcohol as described by the writer in 1946, and confirmed recently by Freeman and Heimberger (1948), Shelden and Bors (1948) and Gingras (1948), has proved very successful indeed in transforming spastic paralysis into a flaccid type which, from the point of view of rehabilitation, is so much more manageable. This method has superseded radical operations, such as posterior rhizotomy (Foerster 1929) and anterior rhizotomy (Munro 1945), which necessitate general anaesthesia, laminectomy, and all the other discomforts and dangers which inevitably are associated with major operations.

REFERENCES

CUTLER, C. W. (1945): Journal of the American Medical Association, **129,** 153.

DICK, T. B. (1949): " Rehabilitation in chronic paraplegia "—M.D. Thesis, Manchester University.

ELSBERG, C. (1940): " Injuries of the skull, brain and spinal cord." London: Baillière, Tindall and Cox. 495.

FOERSTER, O. (1929): " Die traumatischen Lasionen des Rückenmarks." Handbuch der Neurologie, Bergründet von M. Lewandowsky. Berlin: J. Springer, **2,** 1721.

FREEMAN, C. W., and HEIMBERGER, R. F. (1948): Journal of Neurosurgery, **5,** 556.

GINGRAS, G. (1948): Bulletin of the Canadian Veteran Administration.

GOWLLAND, E. L. (1934): Medical Press, **188,** 81.

GOWLLAND, E. L. (1941): British Medical Journal, **1,** 814.

GUTTMANN, L. (1945): Medical Times. New York, **73,** 318.

GUTTMANN, L. (1946): British Journal of Physical Medicine, N.S. **9, 130,** 162.

GUTTMANN, L. (1947): Proceedings of the Royal Society of Medicine, **40,** 219. (Section of Neurology, 9.)

GUTTMANN, L., and WHITTERIDGE, D. (1947): Brain, **70,** 361.

GUTTMANN, L. (1948): " Bedsores." British Surgical Practice, **2,** 65. " Management of paralysis." (In the press.) British Surgical Practice. London: Butterworth & Co. (Publishers), Ltd.

HAYNES, W. G. (1946): American Journal of Surgery, N.S. **72,** 424.

MCLEAN, A. J. (1935): North-west Medicine. Seattle. **34,** 84.

MCCRAVEY, A. (1945): Journal of the American Medical Association, **129,** 152.

MUNRO, D. (1945): New England Journal of Medicine, **233,** 453.

RIDDOCH, G. (1927): Proceedings of the Royal Society of Medicine, **21,** 637. (Section of Orthopaedics, 33.)

SANDIFER, P. H., and GUTTMAN, L. (1944): Middlesex Hospital Journal, **44,** 67.

SHELDEN, C. H., and BORS, E. (1948): Journal of Neurosurgery, **5,** 385.

HINDQUARTER AMPUTATION

H. A. BRITTAIN, NORWICH, ENGLAND

Gordon-Taylor described interinnomino-abdominal or hindquarter amputation as " one of the most colossal mutilations practised on the human frame." In 1934, in collaboration with Wiles, he reviewed all cases reported in the literature and showed that the mortality rate had been 60 per cent. In 1939, he reported eleven personal cases. Since that time the operation has been practised with increasing success. The first reports in American literature were in 1942 (Leighton) and 1943 (King and Steelquist). More recently twelve hindquarter amputations were reported by Beck and Bickel (1948) but curiously without any reference at all to the pioneer work of Gordon-Taylor whose personal series now totals thirty-three cases.

The operation is one of great magnitude and should be undertaken only after careful deliberation. There are still many surgeons who doubt whether it is ever justified, their doubts being based not only on the slender chances of survival of patients whose hindquarters are amputated for malignant disease but also on the assumption that existence after such amputation must of necessity be miserable. It is the purpose of this paper, in recording five cases, to show that greater optimism may be justified and that the joys of life can still be indulged in despite the mutilation. It has even been possible to fit these patients with artificial limbs and teach them to walk without crutches. So far as is known this has not been recorded before.*

FIG. 1

Case 1. W. L. Ossifying chondroma of the ilium, potentially malignant, reaching from the lower pole of the left kidney to the left hip joint. It was increasing steadily in size and, after a period of observation, hindquarter amputation was clearly inevitable.

CASE REPORTS

Case 1. W. L., aged 42 years—First attended in 1938 with one year's history of swelling in the left iliac region extending to the hip-joint and the loin, steadily increasing in size. Radiographs suggested an ossifying chondroma of the ilium (Fig. 1). Biopsy supported the diagnosis of non-malignant ossifying chondroma. After one year the tumour was undoubtedly larger and was reaching up to the lower pole of the left kidney. In 1940 the patient had lost weight and did not look well. There was still no evidence of secondaries in the chest or elsewhere. After consultation with Sir Harry Platt, and examination of biopsy specimens by Professor Baker, it was agreed that hindquarter amputation was justified. This was done in June 1941. The operation was complicated by a urinary fistula which healed six months later. He is now alive and well, and has no complaints. He uses crutches and has adapted his life accordingly, and is unwilling to consider the fitting of an artificial limb.

Case 2. F. G., aged 26 years—First seen October 1945 when she gave a six months' history of a painful lump on the lateral aspect of the right thigh, increasing rapidly in size. Clinical examination showed a tumour of the right femur in the region of the greater trochanter. Open biopsy proved that it was a spindle-cell sarcoma (Fig. 2). Clinical and radiographic examination showed no evidence of secondary growths. Hindquarter amputation was performed December 1945. There was some sloughing of the upper part of the flap and oedema of the perineum but convalescence was otherwise uneventful (Figs. 5–7). More skin than usual had been left in the posterior flap in the hope that it might prove possible to fit an artificial limb. A limb was fitted six months later in 1946. The patient is still alive and well, and three and a half years after amputation is walking happily without crutches (Fig. 8).

* *This paper was submitted for publication October* 16, 1948. (*See Editor's footnote at the end of the article.*) *It was read to the Boston Orthopaedic Club, January* 1949.

Case 3. A. B., aged 42 years—First attended in July 1947. Clinical examination showed Von Recklinghausen's neuro-fibromatosis. Complained-of a painful lump on the antero-lateral aspect of the left thigh. During the previous two months the tumour had grown with increasing speed and begun to ulcerate. Biopsy showed evidence of an undifferentiated spindle cell sarcoma (Fig. 3). There was no clinical or radiographic evidence of secondary deposits. After hindquarter amputation convalescence was uneventful but three months later the patient died from massive secondary growths in the lungs.

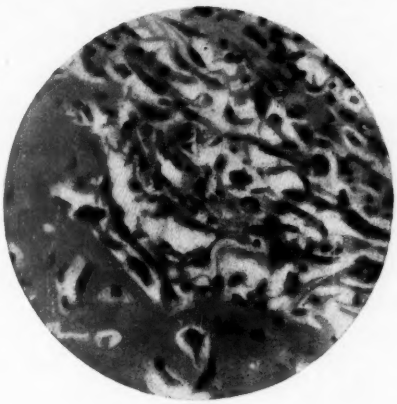


FIG. 2

Case 2. F. G. Spindle cell sarcoma—mass of tumour cells exhibiting pleomorphism; main cell type spindle; some giant and multinucliate forms; on the left side pink-staining homogeneous mass of osteoid tissue.

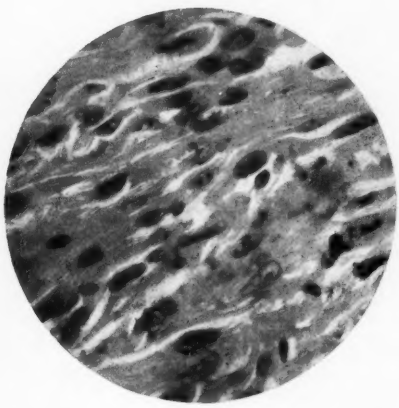


FIG. 3

Case 3. A. B. Spindle-cell sarcoma—closely packed mass of spindle-shaped cells; nuclei show alteration in size. Numerous mitotic figures and pleomorphism was seen in other sections.

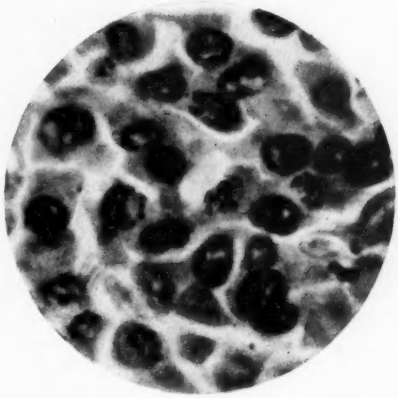


FIG. 4

Case 4. G. S. Plasmocytoma—sheets of closely packed cells, some oval, some polygonal; characteristic appearance of plasma cells; nuclei most eccentric with chromatin in darkly staining clumps.

Case 4. G. S., aged 34 years—First seen July 18, 1947, when he gave a history of having slipped and strained his hip one week earlier. Radiographs showed a tumour of the upper end of the femur involving the greater trochanter with fracture through the lesser trochanter which was avulsed; the appearances suggested an osteoclastoma. Investigation of the family history showed that one sister died at the age of twenty-eight years from carcinoma of the breast, and another sister died at the age of thirty-six years from carcinoma of the uterus. After open biopsy it was reported that the tumour was a plasma-celled myeloma (plasmocytoma) (Fig. 4). After consultation with other pathologists and Mr Osmond-Clarke,

FIG. 5

FIG. 6

Case 2. Patient with hindquarter amputation who walked with an artificial limb without crutches. Figure 6 shows the prosthesis, the socket being made to a cast of the stump and fitted with an automatic hip lock.

FIG. 7

Case 2. Radiograph after hindquarter amputation showing that no more than a small fragment of ilium remains. She walked with an artificial limb without crutches, weight-bearing being from the costal margin.

hindquarter amputation was performed on December 5, 1947. There was pyrexia for some days but recovery was otherwise uneventful and the patient is still alive and well. He has been fitted with a prosthesis and is now walking and has discarded his crutches.

Case 5. G. W., aged 25 years—History of tuberculosis of the right hip joint with intermittent activity of disease since the age of five years. Three years ago ischio-femoral arthrodesis was attempted but failed owing to involvement of the graft by disease. The hip was unstable and there was four inches of true shortening. Many sinuses were discharging, two of them profusely. Radiographs showed extensive disease of both ilium and ischium with sequestration of the ischio-femoral graft. The patient's general condition was poor: there was a swinging temperature from 101 to 103 degrees; weight had gone down from eleven stone (154 pounds) to eight and a half stone (120 pounds); there had been many attacks of diarrhoea; the Congo red test for amyloid disease was positive; radiographs of the chest showed an early active lesion in the right lung; haemoglobin was 52 per cent. After repeated blood transfusions the haemoglobin level rose to 82 per cent., and under the control of streptomycin, trans-iliac amputation was performed. Four pints of blood were transfused during and immediately after the operation. Nine months later the patient is alive and well.

Technique of operation—The operative technique described by Gordon-Taylor has been followed faithfully except that in Case 2 the common iliac artery was tied instead of the external iliac artery. This certainly made the operation easier but there was some sloughing in the anterior part of the flap. It is difficult to attribute sloughing of the *anterior* flap to such arterial ligation but nevertheless the possibility of massive sloughing after ligation of the common iliac artery must be recognised. The only other divergence from the technique originally described was that the ilium was cut through in a few seconds with hammer and chisel instead of with a Gigli saw. In the five cases here reported the operative time was ninety minutes, eighty minutes, eighty-two minutes, seventy-five minutes and sixty-five minutes respectively. It is interesting to compare these times with Gordon-Taylor's average time of sixty-five minutes and also with the four hours and forty minutes recorded by King and Steelquist for trans-iliac amputation. The importance of blood transfusion throughout the operation, as in all such major procedures, calls for no emphasis. Case 1 illustrates the danger of urinary fistula. It is believed that the injury in that patient was inflicted on the bladder at the time that the symphysis pubis was being divided. Special care must of course be taken to avoid damage to the ureters.

THE FITTING OF ARTIFICIAL LIMBS TO HINDQUARTER AMPUTATIONS

In the second case reported in this series, more skin than usual was left in the posterior flap in the hope that some form of prosthesis might be fitted. At that time, in 1946, the limb-fitting surgeons at Queen Mary's Hospital, Roehampton, thought that the fitting of an artificial limb was impracticable; but after consultation with Mr F. H. Powley of the Cambridge branch of Messrs Hanger & Co., Ltd., a limb was fitted to this young widow who was determined to persevere despite the almost insuperable difficulties.

Mr Powley writes: "The latest type of artificial limb that has been supplied for disarticulation at the hip joint was fitted in this case, there being just enough hindquarter left to which a socket could be fitted with the necessary seating. A very careful cast was made of the pelvic stump on which the socket was made. The limb was so fitted that there was minimal movement as the patient propelled and rotated her pelvis in taking a forward step. The artificial limb attached to the socket was of light metal and had the latest improvements in mechanical detail and design, including an automatic hip lock. This kept the thigh rigid with the socket while the patient was walking; but by pressing a small button over the outer side of the hip joint she could tilt the socket forward and sit quite normally. On rising to walk, the hip lock engaged automatically. Success in this case was due largely to the determination of the patient by which she succeeded in making the fullest possible use of the artificial limb."

FIG. 8

Case 2. Reproduction from a ciné film showing patient walking with prosthesis, without crutches, after hindquarter amputation.

Three and a half years later this patient reports that she is well, still wearing her artificial limb and attending to all household duties. She can walk half a mile without fatigue or pain (Fig. 8). It may well be asked: " On what is she walking? " We are aware of tibial-bearing in below-knee amputations and of ischial-bearing in below-knee and above-knee amputations. We recognise that it is possible to bear weight after disarticulation through the hip joint. But where is weight borne after hindquarter amputation? Radiographs show that in this particular case a small fragment of ilium remains lateral to the sacro-iliac joint but this does not provide a weight-bearing area (Fig. 7). There can be little doubt that most of the weight is taken on the ribs and lower thorax. As the patient takes weight on the artificial limb she inspires reflexly, holds her breath, and, being a thoracic-breather, thus fills the bucket of the limb.

This determined young woman was the first patient to my knowledge to walk without crutches on an artificial limb after hindquarter amputation. Others, in this small series of cases, have followed her example. There seems no reason why every patient who suffers hindquarter amputation should not learn to walk without crutches.

REFERENCES

BECK, N. R., and BICKEL, W. H. (1948): Interinnomino-Abdominal Amputations: Report of Twelve Cases. Journal of Bone and Joint Surgery, **30 A,** 201.

GHORMLEY, R. K., HENDERSON, M. S., and LIPSCOMB (1944): Interinnomino-Abdominal Amputation for Chondro-sarcoma and Extensive Chondroma: Report of Two Cases. Proceedings of Staff Meeting, Mayo Clinic, **19,** 193.

GORDON-TAYLOR, G., and WILES, P. (1934): Interinnomino-Abdominal (Hindquarter) Amputation. British Journal of Surgery, **22,** 671.

GORDON-TAYLOR, G. (1939): A Further Review of the Interinnomino-Abdominal Operation: Eleven Personal Cases. British Journal of Surgery, **27,** 643.

KING, DON., and STEELQUIST, J. (1943): Trans-iliac Amputation. Journal Bone and Joint Surgery, **25,** 351.

LEIGHTON, W. E. (1942): Interpelvi-abdominal Amputation: Report of Three Cases: Archives of Surgery, **45,** 913,

Editor's Note—This paper was received for publication in October 1948. Since then other examples have been reported of patients who have been fitted with artificial limbs and have learned to walk without crutches after hindquarter amputation (Mitchell and Baird: British Medical Journal, November 1948, **2,** 940; Wise, R. A.: Journal of Bone and Joint Surgery, April 1949, **31 A,** 426).

PULSATING ANGIO-ENDOTHELIOMA OF THE INNOMINATE BONE TREATED BY HINDQUARTER AMPUTATION

GORDON GORDON-TAYLOR and PHILIP WILES, LONDON, ENGLAND

From the Surgical Unit; Middlesex Hospital, London.

The infrequency of pulsating angio-endothelioma of bone appears to merit the publication of an individual case in a short note rather than its inclusion in a list of thirty-three personal interinnomino-abdominal (hindquarter) amputations wherein the case might more readily be overlooked. An appeal to the classical work of Ewing (1942) vouchsafes the information that the growth of endotheliomata tends to be slow but progressive, and thus they are comparatively benign. More particularly, by comparison with the carcinomata and sarcomata which they may resemble histologically, the course of endotheliomata is relatively favourable. When undisturbed by the knife the growth capacity of some of these tumours seems to be confined within certain limits which do not apply to carcinoma and sarcoma.

It is said that endotheliomata spread by local infiltration, principally along existing channels such as lymph vessels and spaces, while extension to neighbouring lymph nodes, if it occurs at all, is late. Such infiltration accounts for the difficulty of thorough extirpation and for the frequency of persistent local recurrence, each recurrence being apt to show an increasing capacity for growth. This fact encourages the belief that the appropriate treatment is complete removal of the organ involved and not merely local extirpation of the tumour.

Endothelioma of bone tends to replace pre-existing tissues with little or no increase in the size of the bone, but with perforations. Three types of bone endothelioma were distinguished by Ewing: 1) solitary, bulky telangiectatic angio-endothelioma; 2) multiple endothelioma of bone; 3) diffuse endothelioma of bone. The solitary angio-endothelioma of bone, which is the type of tumour present in the patient now reported, affects adults and principally involves the long bones. It grows steadily, perforates and invades the soft tissues and may, as a late feature, metastasize to the lungs; it may pulsate and produce a bruit. Considerable variation in size may be observed; it is usually painful and sometimes causes pathological fracture. The tumour consists of masses of large cuboidal cells in cords or pseudoalveoli enclosing freely circulating blood.

Ewing also reminds us that a somewhat similar clinical and histological picture may be given by metastatic carcinoma of renal or adrenal origin, and that such a primary tumour must always be excluded in formulating the diagnosis.

Other pulsatile osteolytic tumours may occur in bone, such as telangiectatic osteogenic sarcoma and giant-cell tumour. Telangiectatic osteogenic sarcoma usually affects children and is characterised by exceedingly rapid growth and early pulmonary metastases. Giant-cell tumour of bone, when vascular and pulsatile, may cause great difficulty in differential diagnosis, its growth being slow like the angio-endothelioma; but usually the periosteum lays down a bony capsule which gives rise to characteristic radiographic appearances.

Case Report—E. B., female, 42 years of age, was admitted to hospital on April 15, 1949, with a history that five months earlier she had noticed pain in the right hip, rather posteriorly, which persisted for a month. The pain was a dull ache, with an occasional sharper element, gradually spreading down the front of the thigh to the knee and from there to the front of the shin and the toes. In the lower leg it tended to be a shooting pain, rather sharp like a knife. It came on particularly in the morning and was eased by rest. Lying on the right side caused discomfort in the limb which prevented her from getting to sleep, but did not awaken her when she was once asleep. The pain made her limp.

On examination—There was a mass in the right iliac fossa exhibiting marked expansile pulsation which could be obliterated by compression of the aorta. A to-and-fro murmur

could be heard over the tumour. The tumour presented on both surfaces of the iliac part of the os innominatum. The diagnosis seemed to lie between a tumour of the innominate bone and an iliac aneurysm, with the odds heavily on a bone tumour. The radiograph showed destruction of the wing of the ilium (Fig. 1).

There was nothing abnormal in the cardiovascular, respiratory or central nervous systems. The appetite had been indifferent but there was no other abnormality in the alimentary system. The urinary system was normal and menstruation had ceased for the last eight months. The patient was rather pale and thin; the skin, hair and nails were normal; thyroid not palpable; no superficial glands felt. Pulse rate 76; regular; volume and tension normal : blood pressure 160/85 : heart sounds normal.

FIG. 1

Radiograph showing destruction of the ala of the right ilium by angio-endotheliomatous tumour. The pyelogram shows a defect in the ureter at the pelvi-rectal junction on the right side.

Blood count (*February* 1, 1949)—Haemoglobin 68 per cent.; red blood corpuscles 3·85μ; colour index 0·88; P.C.V. 34; white blood corpuscles 3,600 (polymorphonuclears 63 per cent., lymphocytes 33 per cent., monocytes 4 per cent.) hypochromia; microcytosis; anisocytosis. Erythrocyte sedimentation rate 20 mm. in one hour.

Faeces (*February* 8, 1949)—Occult blood—Benzidine, faintly positive; Guaiacum, negative. *Radiographs of skull, ribs, sternum and vertebrae* (*February* 8, 1949)—No evidence of myelomatosis in the ribs, skull, sternum or spine. The opacity in the pelvis was presumed to be a solitary plasma cell tumour.

Blood examination (*February* 11, 1949)—Total protein, 7·0 gms. per 100 ml.; albumen-globulin ratio, 2·3 : 1; fibrinogen, 0·38 gms. per 100 ml.

Red bone marrow examination (*February* 21, 1949)—Total nucleated cell count 67,000 c.mm.; myeloid/erythroid ratio 3 : 1; proerythroblast 2 per cent.; early normoblast 4; intermediate normoblast 7; late normoblast 17; megalokaryocyte 0·2 per cent.; myeloblast 1; premyelocyte 3; myelocyte 9; metamyelocyte 17; polymorphonuclear 33; lymphocyte 5 per cent.; monocyte 1 per cent.; plasma cell 0·3 per cent.; no myeloma cells seen.

Intravenous pyelography—Preliminary films—The tumour in the right ilium was considerably larger than on previous radiographic examination. Both kidneys excreted the dye at five minutes. The renal drainage system on the left side was normal. On the right side there was a constant defect in the ureter at the pelvi-rectal junction (Fig. 1). The pelvis and calyces on this side were otherwise normal. The nature of the defect was obscure and the lower part of the right ureter was displaced medially as if by the tumour. An impression was also present on the right side of the bladder. Retrograde pyelogram showed the same appearances as in the intravenous pyelogram. The narrowing at the pelvi-ureteric junction could be explained by extrinsic pressure, and the deviation of the lower end of the ureter by the mass. There did not appear to be any clear evidence of hypernephroma.

FIG. 2

FIG. 3

Specimen of angio-endothelioma of innominate bone showing relation to the iliac vessels and displacement of the ilio-psoas (Fig. 2) and the same specimen on section (Fig. 3).

Biopsy of tumour of right ilium (*March* 17, 1949)—An incision was made half an inch below the anterior part of the iliac crest and parallel to it. There was considerable bleeding from the subcutaneous tissue and below the deep fascia; in the muscle the bleeding became so profuse that further approach from this direction was impossible. The iliac crest was exposed and the muscle stripped off it for three-quarters of an inch. Two pieces of bone were removed for biopsy: they were macroscopically normal. There was by this time so much bleeding from the bone as well as the other tissues that further progress was not practicable. Accordingly, the bleeding was controlled by Oxycel gauze swabs and the wound closed, leaving two of these inside. The tumour could be felt as a hard mass beneath the muscle, but it was not exposed or incised. Histological examination of the biopsy fragments showed normal bone tissue, no tumour tissue being present.

Radiograph of chest (*March* 17, 1949)—There was some cardiac enlargement and two opacities in the right lower thoracic zone. The appearances indicated old calcified tuberculous foci. No secondary deposits seen.

Operation (*April* 20, 1949)—The usual preliminary operative approach to a hindquarter amputation was employed, which also gave adequate approach to the iliac vessels. Owing to the extent of the tumour the whole innominate bone required removal; disarticulation at the sacro-iliac joint was necessary (Figs. 2–3). During the operation and subsequent post-operative

period six pints of blood were transfused. The patient made an uneventful recovery from the hindquarter amputation and left hospital, June 9, 1949, walking well on crutches.

Histological Report (*April* 20, 1949)—Tumour 4 cms. × 3 cms. × 3 cms. eroding the centre of the iliac bone. The mass appeared to have a fibrous capsule; it had not invaded but had displaced the iliacus muscle above and the gluteus below. On section, the tumour was composed of a firm outer rim three-quarters of an inch wide with a bony vascular centre:

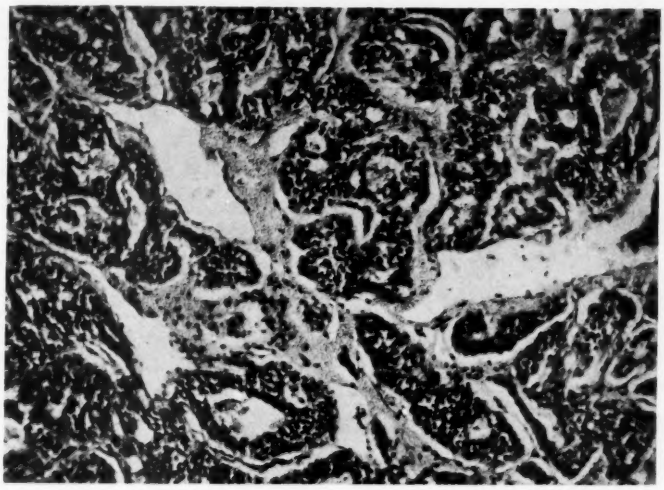


FIG. 4

Microscopic section of haemangio-endothelioma of the innominate bone.

there were many spicules of bone in the tumour. Section showed a cellular tumour replacing the bone: the tumour showed numerous spaces containing blood, surrounded by several layers of endothelial cells. The degree of differentiation indicated a moderate degree of malignancy (Fig. 4).

REFERENCE

EWING, J. (1942): Neoplastic Diseases. Philadelphia and London: W. B. Saunders & Co., **342**, 360.

THE PAINFUL SHOULDER

Review of One Hundred Personal Cases with Remarks on the Pathology

R. J. W. WITHERS, BELFAST, NORTHERN IRELAND

From the Orthopaedic and Fracture Service, Royal Victoria Hospital, Belfast *

In considering the well-defined group of conditions in which there is limitation o alteration in the range of movement of the shoulder joint, pain on attempted movement and sometimes muscle wasting, with no radiographic evidence of abnormality, certai anatomical features must be recalled. In the adult the capsule is fused with the overlyin short rotator muscles; whereas in infants the capsule is separated from the deep muscles o the shoulder by areolar tissue, the tendons being short and the muscle fibres extending t within a few millimetres of their humeral attachment. With increasing age the tendon become longer and in old age the supraspinatus tendon may extend as far proximally as th suprascapular notch. The degree of adhesion of the tendons to the capsule increases with age, and in older subjects the fusion is complete. The changes are more marked in the upper part of the capsule so that in adult life the entire breadth of the supraspinatus tendon, and the upper parts of the infraspinatus and subscapularis tendons, are intimately fused with the shoulder capsule.

The frequency with which rupture of the supraspinatus tendon is found at post-mortem examination was emphasized by Codman (1931) and Codman and Akerson (1931). Grant and Smith (1948) demonstrated that degenerative changes in the capsule, especially in its upper part, increased in arithmetical progression as age advanced. In ninety-five dissecting-room specimens they found degenerative changes causing complete or incomplete rupture of the capsule in no case of a subject aged 17 to 40 years; in 25 per cent. of subjects aged 40 to 56 years; in 39 per cent. of subjects aged 56 to 76 years; and in 50 per cent. of subjects aged 76 to 86 years. It must also be recognised that falls on the shoulder are more likely to cause injury to the upper part of the capsule than to its anterior or posterior aspects. For these three reasons—fusion of the supraspinatus with the capsule, increasing degeneration of this fused part as years advance, and liability to direct injury from falls—it is evident that most cases of " painful shoulder " are due to pathological changes in the upper part of the capsule.

In this review, a series of one hundred patients have been re-examined at intervals ranging from one to three years after cessation of treatment. The series includes only patients in whom there was no radiographic evidence of abnormality; examples of calcareous deposit in the tendons round the joint have been excluded. The average age was fifty-two years. There were fifty-two men and forty-eight women. The right shoulder was affected in fifty-seven and the left in forty-three patients. There was a definite history of injury in 60 per cent. Three clinical groups were differentiated: 1) rupture of the supraspinatus; 2) supraspinatus tendinitis; 3) capsulitis of the shoulder.

Rupture of the supraspinatus—After a fall on the shoulder there was diffuse pain over the shoulder and sometimes pain referred to the insertion of the deltoid muscle, with inability to elevate the limb, and inability to support it in the elevated position after it had been so placed, but with a normal range of passive movement. In this group the disability was due to loss of the power of fixation of the humeral head to the glenoid by the spinati; the deltoid was thereby put at such a disadvantage that although it was able to contract, as could be felt by the examining hand, it was unable to sustain true shoulder movement. In cases of recent rupture this loss of fixator function may be no more than a reflex phenomenon

* *Paper read at the Belfast meeting of the British Orthopaedic Association, October* 21, 1948.

induced by pain, because the uninjured infraspinatus, which is a larger and more important tensor and depressor of the humeral head than the supraspinatus, is also thrown out of action. If this is accepted, it follows that it is impossible on clinical grounds to distinguish between complete and incomplete ruptures. Clinical and operative experience has shown that this is true.

In establishing the diagnosis there is a place for arthrography. Radiographs are taken after the injection of 6 to 8 cubic centimetres of perabrodil (35 per cent. solution) either into the shoulder joint as suggested by Oliver Axen (1941) or into the subdeltoid bursa as practised by the writer. It can then be demonstrated in a few minutes whether or not there is communication between the bursa and the joint cavity. It must be recognised, however, that although demonstration of a communication is strongly suggestive of complete supraspinatus tendon rupture it is not absolutely conclusive because in a small percentage of normal joints there is communication with the bursa (Axen 1941).

Supraspinatus tendinitis—In this series there were twenty-seven cases of supraspinatus tendinitis. After direct injury, or sometimes spontaneously, a painful arc of movement during mid-elevation develops, with weakness of the limb and often with abnormal scapulo-humeral rhythm but without limitation of the range of movement. Symptoms had been present, on the average, for five months. The disability is often ascribed to impingement of an irritated or inflamed supraspinatus tendon against the under-surface of the acromion, but in two cases in which rupture of the supraspinatus tendon had been treated by suture with excision of the acromion there was still the typically persistent pain of supraspinatus tendinitis. So much bone had been excised that acromial pressure could not have been the cause. The work of Inman, Saunders and Abbott (1944) may throw light on this problem. In myographic studies of the shoulder joint they noted that the peak of contraction of the supraspinatus muscle occurred between 80 and 120 degrees elevation of the limb. This muscle contracts most powerfully in that particular phase of elevation which, in the group under consideration, is associated with pain, and it is possible that active contraction of a supraspinatus tendon which is irritable from inflammation or rupture may in itself be sufficient to cause pain.

Capsulitis—Sixty-one cases were classified as examples of capsulitis occurring spontaneously or after injury. There was pain, limitation of scapulo-humeral movement, weakness and muscle wasting. The term capsulitis is used because it is believed that the essential pathology is inflammation of the capsule from injury or irritating causes within the joint. Neviaser (1945) explored ten shoulders in order to determine the underlying pathology and found that the capsule was thickened and adherent to the humeral head; when the joint was manipulated the capsule separated from the head of the humerus in the same way that an adhesive bandage may be torn from the skin; and microscopic section of the capsule showed inflammatory and reparative changes. If this view is accepted it is clear that two stages of the pathological process may be recognised: 1) an early stage of acute inflammation, with inflammatory exudation in and round the capsule and limitation of shoulder movement due to muscle spasm which relaxes under anaesthesia; 2) a later adhesive stage, after resolution of the inflammatory process, in which there are fibrous adhesions limiting movement even when the muscles are relaxed by anaesthesia. This concept of the pathology is borne out clinically, and it is my routine practice to examine every " frozen shoulder " under anaesthesia in order to test the range of movement and put each case into its proper sub-group of irritative or adhesive capsulitis.

TREATMENT AND RESULTS

Rupture of the supraspinatus tendon—Of the twelve patients in this group, five were treated conservatively and seven by operation. *Conservative treatment* consisted in complete rest of the shoulder joint in an abduction splint until the patient was able to raise the limb actively. This took an average period of five weeks. When this stage was reached the splint was

discarded and gentle exercises were practised, at first in the supine position. Three patient regained full painless movement in twelve weeks; in the other two patients three-quarter of the normal range of movement was restored. All returned to full work. *Operative treatmen* was carried out in seven patients, complete rupture of the tendon being found in every case The acromion process was excised and the tendon was repaired. Two patients gained ful recovery of function; two regained almost full movement but with weakness and pain a mid-elevation; and three were not improved at all. The most successful results were secure by simple longitudinal closure of the hiatus without attempting to suture any part of th tendon to the tuberosity.

Supraspinatus tendinitis—Of the twenty-seven patients in this group, thirteen gaine full recovery without any treatment at all, or after simple rest in an abduction splint fo five or six weeks. *Operative treatment* was advised in fourteen patients and carried out i eleven. In each case the acromion process was first excised. When the subdeltoid burs was opened it was found in eight patients that there was a localised L-shaped or longitudina tear of the upper part of the capsule; in two there was swelling and longitudinal splitting of the upper surface of the supraspinatus tendon; and in one there was rupture of the articular surface of the tendon discovered only after longitudinal incision by which to explore the " lump " in it. This group of cases illustrates the difficulty in differentiating clinically between simple tendinitis and actual rupture of the supraspinatus. Of the nine patients with incomplete rupture of the tendon the most successful results were gained in those in whom it was possible to bring the margins together longitudinally without tension. They recovered full painless movement within four months with no more than slight residual weakness when tested against resistance. All patients in whom there was a more complete rupture, and an attempt was made to suture the tendon to the tuberosity, made an incomplete recovery or no recovery at all.

Capsulitis—There were sixty-one patients with capsulitis. All were examined under anaesthesia so that they could be differentiated into the sub-groups of irritative and adhesive capsulitis.

Irritative capsulitis—Twenty patients belonged to this group and were first seen after an average period of seven weeks from the onset of symptoms. Pain was severe and almost constant, and it was worse in bed. Movement was entirely scapulo-thoracic and elevation was to 70 degrees only. Under anaesthesia full movement at the scapulo-humeral joint was possible. Treatment was primarily by rest in an abduction splint or sling, no evidence being found that the splint had any advantage over the sling. Indeed several patients treated in a splint had to discard it because the pain was increased. Gentle exercises were encouraged at an early stage, the exercises first being practised with the patient supine; they were encouraged by heat in its various forms. On the average, full function with a normal range of painless movement was restored within three and a half months, although three patients developed adhesive capsulitis and needed manipulations before recovery was complete.

Adhesive capsulitis—There were forty-one patients in this group who first attended for treatment after an average period of eight months. Pain was less severe, and it was felt only after attempted movement of the shoulder which was mainly scapulo-thoracic. The range of movement was not altered under anaesthesia. When movement was forced, adhesions were felt and heard to tear. In this group the primary treatment was by manipulation under anaesthesia. Since lateral rotation of the humerus is necessary for full abduction, this movement should be carried out first; in most cases this alone was sufficient to free the shoulder. After manipulation the limb was supported in an abduction splint for two weeks and active exercises were practised from the beginning. There was full recovery within a period ranging from two weeks to four months. In five cases three manipulations were necessary; in the other thirty-six cases one manipulation was sufficient. In the follow-up of this group, crepitus was found constantly during passive movements of the shoulder joint but the patient was usually unaware of it and the significance of such crepitation is uncertain.

SUMMARY

1. In a series of one hundred personal cases of " painful shoulder " rupture of the musculotendinous cuff was proved by operation in eighteen cases and was presumed in eight further cases, namely, in about one quarter of the series.

2. Of the eighteen cases treated by operation through the transacromial approach, perfect results were obtained only in four. The other results were unsatisfactory. In five, movement was good but there was pain or weakness of the limb. In nine the condition was unaltered or made worse.

3. Conservative treatment of traumatic lesions of the supraspinatus tendon is therefore advocated. The advisability of rest of the shoulder joint in an abduction splint for five or six weeks is stressed. Operative exposure, through a transacromial approach, is recommended only when conservative measures have failed.

4. When there is rupture of the cuff there is evidence that simple longitudinal suture of the gap after freshening of the margins is likely to give better results than attempts to suture the medial end of the rupture to the greater tuberosity. Most ruptures have a longitudinal extension of the initial transverse tear, and direct suture of the medial edge of the gap to the greater tuberosity may lead to shortening of the tendon and interference with its function.

5. Ruptures of the supraspinatus tendon may cause no symptoms, lead to complete loss of function, or cause a painful arc of movement during mid-elevation. The clinical state depends upon whether or not the condition of the ruptured tendon is causing pain, and whether the other short rotator muscles of the shoulder can compensate in tensor and fixator actions for the loss of action of the supraspinatus.

6. Capsulitis is a convenient term by which to describe inflammatory lesions of the capsule and bursae around the shoulder joint. The initial stage of irritative capsulitis may develop to the later stage of adhesive capsulitis. Differentiation is important because treatment in the first stage is by rest, and in the second by manipulation and exercise.

7. The subdeltoid bursa is the " peritoneum " of the spinatus tendons; like the peritoneum it shares the pathology of the organs it protects and is itself seldom the site of primary pathological processes.

8. The management of the " frozen shoulder," whether loss of movement is protective or adhesive, calls for time and patience but the ultimate outlook is good.

REFERENCES

ARMSTRONG, J. R. (1947): Lancet, **1,** 94.
AXEN, O. (1941): Acta Radiologica, **22,** 258.
BOSWORTH, D. M. (1940): Journal of Bone and Joint Surgery, **22,** 369.
BURNS, B. H., and ELLIS, V. H. (1937): Recent Advances in Orthopaedics. London: Churchill, 151.
CODMAN, E. A., and AKERSON, I. B. (1931): Annals of Surgery, **93,** 348.
CODMAN, E. A. (1931): Surgery, Gynecology and Obstetrics, **52,** 579.
GRANT, J. C. B., and SMITH, C. G. (1948): Anatomical Record, **100,** 666.
HARRISON, T. G. (1948): Personal communication on Dissections of the Shoulder Joint.
HEY GROVES, E. W., and GRIFFITHS, S. J. (1932): Medical Annual. Bristol: John Wright & Sons, Ltd., 483.
INMAN, V. T., SAUNDERS, J. B. de C., and ABBOTT, LE ROY (1944): Journal of Bone and Joint Surgery, **26,** 1.
McLAUGHLIN, HARRISON L. (1944): Journal of Bone and Joint Surgery, **26,** 31.
MERCER, W. (1943): Orthopaedic Surgery. 3rd Edition. London: Edward Arnold & Co., 667.
NEVIASER, J. S. (1945): Journal of Bone and Joint Surgery, **27,** 211.
WATKINS, J. T. (1925): Journal of Bone and Joint Surgery, **7,** 787.
WILSON, P. D. (1931): Journal of the American Medical Association, **96,** 433.
WITHERS, R. J. W. (1942): Ulster Medical Journal, **11,** 1.

THE PAINFUL SHOULDER

Significance of Radiographic Changes in the Upper End of the Humerus

STEWART H. HARRISON, BIRMINGHAM, ENGLAND *

Radiographic changes in the upper end of the humerus at the site of attachment of the musculo-tendinous cuff have been described by Codman (1934) and Lindblom and Palmer (1939). The literature is concerned mainly, however, with bone changes detected after complete tears of the cuff whereas the intention of this paper is to emphasize the importance of the early bone changes that may occur *before there is clinical evidence of rupture of the tendons*. These studies show clear evidence of pathological degeneration in and around the shoulder joint, involving bone and soft tissue, which has an important bearing on the diagnosis and prognosis.

The early stage of this degeneration can exist without symptoms, but injury to a joint so affected readily gives rise to the various types of painful shoulder. The term " degenerative lesion " is used (in cases in which there is no clinical evidence of actual rupture of the musculo-tendinous cuff) because the radiographic changes to be demonstrated are known to be associated with degeneration in the overlying tissues and there is no evidence of an inflammatory process such as might be indicated by the term " tendinitis."

Pathology and radiographic features—The characteristic pathological features are well illustrated by reference to an actual case (Figs. 1–6). The subject was a male aged thirty-six years who died from head injuries. Relatives stated that at no time had he complained of injury or of symptoms referred to the shoulder. Post-mortem examination showed evidence of an old tear of the subscapularis muscle (Fig. 1). On the deep aspect of the tendon there was an area, about $1 \times 1{\cdot}5$ centimetres, which had lost its normal sheen and had a roughened surface (Fig. 2). The tendon was bound down to the anatomical neck by adhesions which made reflection difficult, and the underlying part of the head of the humerus showed pitting and erosion of cartilage. Radiographs of the specimen showed a well-defined area of cystic cavitation (Fig. 4). The affected area of bone and tendon was sectioned and the appearance of the cyst is illustrated in Figure 5. The cyst was multilocular and lined by fibrous tissue; and the adjacent area of bone and soft tissue had an appearance of homogeneous degeneration. A radiograph of this section suggested that there was communication between the cyst and the anatomical neck at the site of attachment of the supraspinatus (Fig. 6).

Examination of the left shoulder showed a similar early degenerative process affecting the deep surfaces of both supraspinatus and subscapularis (Fig. 3). It is interesting to note that, as in the right shoulder, the changes were more advanced in the subscapularis than in the supraspinatus, though they had not progressed to the stage of tendon rupture. There was adhesion formation, pitting and erosion of articular cartilage, and radiographic evidence of cystic cavitation.

The association of complete tears, incomplete tears, early degeneration, erosion of cartilage and cystic cavitation has been demonstrated in other post-mortem specimens, and there can be little doubt that a degenerative process involving bone and soft tissue is frequently found in the middle and later decades of life. Occupational trauma is undoubtedly a causative factor. This degenerative process is of primary importance in the causation of complete and incomplete tears of the musculo-tendinous cuff.

Radiographic appearances—The characteristic radiographic appearances, shown in Figures 7 and 8, are in marked contrast with those of a normal shoulder in which the clearly defined outline of the head of the humerus and the greater tuberosity and anatomical neck, and the trabeculation of cancellous bone, are constant features (Fig. 9). The changes that indicate

* *Paper read at the Annual Meeting of the British Orthopaedic Association, Belfast,* 1948.

FIG. 1

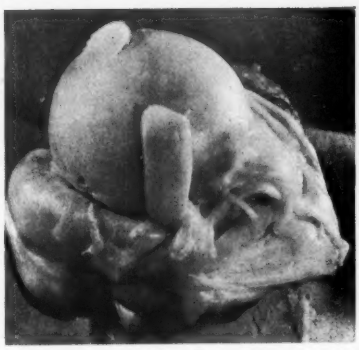


FIG. 2

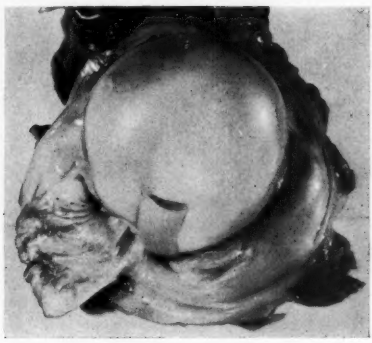


FIG. 3

Case 1. Male, aged thirty-six years. Degenerative lesions of both shoulder joints with no symptoms during life. Specimens removed after death. The right shoulder shows an old tear of the supraspinatus tendon near its insertion (Fig. 1). The deep surface of the capsule shows an area of roughening on the supraspinatus tendon (Fig. 2). The left shoulder shows similar degenerative changes affecting the deep aspect of the subscapularis and supraspinatus (Fig. 3).

FIG. 4

FIG. 5

FIG. 6

Case 1. Radiograph of the dissected right shoulder shows an area of cystic cavitation (Fig. 4). This is seen in macroscopic section in Figure 5, and a radiograph of the section is shown in Figure 6.

a degenerative lesion at the attachment of the musculo-tendinous cuff are: irregularity of the cortical bone of the greater tuberosity; erosion of bone of the articular surface of the head of the humerus; loss of the normal outline of the anatomical neck; cystic cavitation; and areas of sclerosis.

Clinical features—Immediately after injury it is impossible to differentiate clinically between a degenerative lesion and an incomplete tear of the musculo-tendinous cuff, and it is probable that in many cases both exist in the same patient. Shoulders that have sustained injury and show radiographic evidence of a degenerative lesion give rise to a characteristic clinical syndrome with localised pain, limitation of abduction, pain on abduction, a painful arc of abduction, referred pain, nocturnal pain, abnormal scapulo-humeral rhythm and wasting of the spinati.

FIG. 7

FIG. 8

FIG. 9

The radiographic changes characteristic of a degenerative lesion are shown in Figures 7 and 8. They contrast markedly with the appearances in a normal shoulder shown in Figure 9.

The painful arc of abduction has been described as peculiar to supraspinatus tendinitis, but clinical observation suggests that it is found in a wide range of shoulder joint lesions and of injuries and osteoarthritis of the acromio-clavicular joint. Pain referred to the insertion of the deltoid, or to the elbow and wrist joint, is a common finding, and in many patients there is no complaint of pain in the shoulder joint itself. In the differential diagnosis it is often difficult to distinguish abnormalities of the acromio-clavicular joint but pain from this joint is usually referred along the trapezius muscle to the posterior aspect of the ear. Nocturnal pain is a frequent symptom and is an indication that the muscles are reacting by spasm to an irritative process. Abnormal scapulo-humeral rhythm is a further indication of spasm in the muscles guarding the shoulder joint. Wasting of the spinati is rapid in onset and is important from the point of view of treatment. Loss of power of these muscles not only causes impairment of external rotation but also of abduction movement, and when external rotation has once been lost and secondary changes have taken place in the anterior part of the capsule and subscapularis, it is difficult to gain full recovery.

Review of clinical material—An investigation has been made of 145 shoulder joint injuries, excluding major fractures and dislocations. All patients reported with a history of injury and with varying degrees of pain and limitation of movement. Patients diagnosed as having sustained " contusion " were those in whom the radiographic appearances were normal and there was no clinical evidence of specific soft tissue injury; patients diagnosed as having a " degenerative lesion " were those in whom there were radiographic changes at the site of attachment of the musculo-tendinous cuff. All age groups are included and the classification is shown in Table I.

TABLE I

CLASSIFICATION OF 145 CASES OF SHOULDER PAIN AFTER INJURY, WITH INCIDENCE OF MAIN SYMPTOMS

Diagnosis	Total	Pain on abduction	Limitation of movement	Painful arc of movement	Localised pain	Reversed scapulo-humeral rhythm	Nocturnal pain	Referred pain
Degenerative lesions	26	26	22	7	22	3	3	8
Contusions	96	68	74	9	41	3	6	5
Supraspinatus tears	4	4	4		4			
Avulsion of bone	4	4	2		4		2	2
Fractures of tuberosity	3	3	2	1	3			
Calcification supraspinatus	2	1	1		1			
Muscle strain	3	3	2					
Osteoarthritis A.C. joint	2	1	2					
Injury acromio clavicular joint	2	1	2					
Subdeltoid bursitis	2	2	2	2				
Incomplete tear supraspinatus	1	1	1					

TABLE II

ANALYSIS OF ALL CASES OF PAINFUL SHOULDER AFTER INJURY IN PATIENTS OVER FIFTY YEARS OF AGE

	Radiographic findings	Number of Cases	Average duration of treatment
Group 1	Normal appearances	28	4 weeks
Group 2	Degenerative lesion	26	7 weeks
Group 3	Calcified deposit	1	3 weeks
Group 4	(Not radiographed)	15	3 weeks

Radiographs were available in 111 cases and they showed that there was a normal appearance in seventy, a degenerative lesion in twenty-six, acromio-clavicular arthritis in two, fracture of the greater tuberosity in three, calcification of the supraspinatus in two, and avulsion of bone from the greater tuberosity in four. Patients in whom the radiographic appearances were normal and were classified as having sustained "contusions" had an average duration of hospital treatment of eighteen days; those with degenerative lesions had an average duration of hospital treatment of forty-nine days. Four had complete tears of the supraspinatus, confirmed at operation. Of the twenty-six patients with degenerative lesions, fourteen were women and twelve men; and the average age was fifty-two years. This high age group is partly coincidental, because these lesions have been demonstrated

radiographically *in vivo*, and at post-mortem, in patients as young as thirty-six years. Analysis of all patients over the age of fifty years (Table II) indicates that radiographic evidence of degenerative changes was present in about half of them.

At follow-up review, six to twelve months after injury, the significant finding was that every patient with radiographic evidence of degenerative changes reported residual disability. In contrast, apart from one patient who complained of some creaking, no case diagnosed as " simple contusion " showed any residual sign or symptom.

Treatment—This paper is concerned primarily with diagnosis, but two points in treatment justify comment. In the first place, in shoulders that are subject to early degenerative lesions, any movement of the limb that initiates pain may aggravate the symptoms and prolong the disability. To avoid this, one of three courses may be adopted: namely—rest in a sling for six weeks with the limb adducted; elevation and fixation by means of a head halter for six weeks; or excision of the acromion. Treatment by simple rest in a sling is supplemented by supervised exercises with the arm adducted; and similarly halter treatment is controlled by supervised exercises in the abducted position. A halter is certainly more effective than a sling, but it is difficult to persuade patients to persevere with this position for an effective period. Rest in a sling with the limb adducted and internally rotated encourages atrophy of the external rotator muscles and may lead to secondary contracture with a risk of permanent limitation of movement. To avoid these difficulties a series of cases with degenerative lesions has been treated by excision of the acromion but sufficient time has not yet elapsed to justify comment.

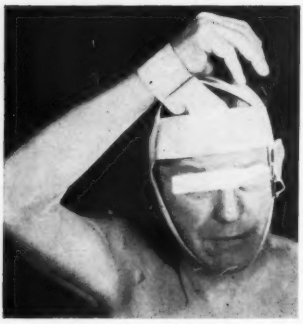


FIG. 10
Head halter with wrist strap.

Secondly, after repair of a complete supraspinatus tear it is important to treat the patient with the limb in abduction. The patient leaves the operating theatre with the arm abducted on a Middledorfs' triangle. The fully abducted and externally rotated position of the arm, necessary for fixation in a head halter, is secured by the third or fourth day (Fig. 10). When the head halter is fitted, the patient is allowed up, the position of abduction being retained for six weeks.

SUMMARY

1. Degenerative lesions of the shoulder joint can often be demonstrated radiographically before there is actual rupture of the musculo-tendinous cuff.

2. The characteristic pathological, clinical and radiographic features of degenerative lesions are described.

3. All injuries of the shoulder joint, however trivial, occurring in patients over middle age, should be studied carefully by radiographic examination.

4. In injuries of the shoulder joint the presence of a degenerative lesion prolongs the duration of symptoms, and the prognosis is less satisfactory than when there is no radiographic evidence of abnormality.

REFERENCES

CODMAN, E. A. (1934): The Shoulder. Boston, Massachusetts. Privately printed.
LINDBLOM, K., and PALMER, I. (1939): Acta Chirurgica Scandinavica, **82**, 133.

EARLY ASSESSMENT OF SUPRASPINATUS TEARS

Procaine Infiltration as a Guide to Treatment

J. TULLOCH BROWN, GLASGOW, SCOTLAND *

From the Accident and Orthopaedic Service, Western Infirmary, Glasgow

One of the difficulties in the management of tears of the musculo-tendinous cuff of the shoulder joint arises from the lack of clearly defined criteria on which to base the indications for operative or conservative treatment early in the course of the disability. Clinical assessment of the extent of the tear is often unreliable and only after the lapse of time does evidence of persistent disability lead to the confident diagnosis of an extensive tear that requires suture. On the other hand, over 50 per cent. of patients with supraspinatus tears may make a satisfactory functional recovery without operation. This paper is concerned solely with the presentation of a simple practical test by which an early decision may be made as to whether or not operative treatment is needed.

A series of 109 cases, diagnosed clinically as supraspinatus tears, was studied with particular reference to the recovery of function obtained without operation and regardless of apparent initial severity. The series included all patients attending the Orthopaedic Clinic of the Western Infirmary, Glasgow, over a period of three and a half years when conservative treatment was employed almost without exception. There were seventy-eight men and thirty-one women, with ages varying from 29 to 72 years (70 per cent. being in the fourth to the sixth decades). Sixty were manual workers. There was a history of precipitating injury or sudden muscle strain in 103 of the 109 cases.

Criteria for diagnosis—The diagnosis of supraspinatus tear was made clinically on the history of sudden pain in the shoulder joint, often referred to the deltoid origin and usually precipitated by a fall or sudden muscular effort, tenderness localised between the acromion process and the great tuberosity, painful arc of abduction movement, loss of voluntary abduction, greater range of passive than of active movement, and radiographic exclusion of fracture, joint lesion or calcified deposit. Tears associated with fractures or dislocations were excluded. Undoubtedly the diagnosis of an incomplete tear cannot be made with certainty on clinical grounds. Cases placed in this group will often include minor contusions and abrasions of the subacromial bursa, contusions of the tendinous cuff and of the tuberosity, as well as true small tears of the tendon substance. Nevertheless the cases in this clinical group, as noted in Table I, showed a uniformly good prognosis and presented no great problem in early treatment. In patients with more acute and more gross symptoms there is less difficulty in reaching a confident diagnosis of shoulder cuff tear, but the prognosis is more uncertain and it is to these more severe lesions that attention is directed.

Clinical assessment—In an endeavour to correlate the initial clinical picture with the ultimate prognosis, cases were graded arbitrarily on the findings recorded at first examination into one of three clinical groups—mild, moderate and severe. " Mild " cases were judged to be those with a characteristically painful arc of movement and with tenderness but with a full range of abduction; " moderate " cases were those in which there was inability to initiate abduction of the arm from the side but ability to sustain the limb in the abducted position after it had been raised passively beyond 90 degrees; in " severe " cases there was both inability to initiate abduction and to sustain the limb in abduction after it had been raised passively.

* *Paper read at the Annual Meeting of the British Orthopaedic Association, Belfast,* 1948.

The patients were treated conservatively by the support of a sling and local heat (79 cases), rest in an abduction frame (18 cases), or repeated injection of local anaesthetic (12 cases). Active shoulder exercises were encouraged as soon as acute discomfort had settled. No particular merit could be ascribed to any one form of treatment and the numbers are too few to justify detailed comparison.

TABLE I
ANALYSIS OF FUNCTIONAL RECOVERY IN 109 PATIENTS TREATED CONSERVATIVELY

Clinical grading	Number of cases	Full recovery	Incomplete recovery	No recovery
Mild	53	46 (87%)	7 (13%)	—
Moderate	22	13 (59%)	7 (32%)	2 (9%)
Severe	34	18 (53%)	12 (35%)	4 (12%)

Results—Analysis of the recovery of function after conservative treatment in the three clinical groups is shown in Table I.

In the group graded clinically as " mild " the prospect of recovery with conservative treatment is good—and mild cases can be segregated clinically with ease. The real problem arises in attempting to determine the best treatment in more severe lesions in which clinical assessment alone is inadequate as a guide to prognosis and treatment. All that it is possible to say is that of cases classed as " severe " about half recover completely without operation even if, at first, it appears that all power of abduction has been lost. In the earlier stages, pain and muscle spasm are such that active abduction is always inhibited. The tendency, therefore, has been to advise operation only if there is not return of abduction power after a period of conservative treatment. The alternative policy of exploring all apparently severe lesions at the outset would lead to many unnecessary operations.

ASSESSMENT WITH PROCAINE INFILTRATION

In a later series of twenty-seven patients, a local anaesthetic was injected into the painful area of the tendon in the hope that relief of shoulder pain and spasm might permit more accurate assessment of supraspinatus function. This investigation was confined to patients who showed complete loss of the power of abduction. Ten cubic centimetres of 1 per cent. procaine was used. After five minutes, the ability to initiate and sustain abduction was reassessed. In a number of patients full active abduction was temporarily restored and these were recorded as showing positive procaine tests. All patients, as in the first series, were observed throughout a period of conservative treatment.

TABLE II
ANALYSIS OF FUNCTIONAL RECOVERY IN TWENTY-SEVEN PATIENTS TESTED INITIALLY WITH PROCAINE

Procaine test	Total cases	Full recovery	Full abduction with residual painful arc	Abduction less than full (to 100 degrees)	No abduction recovery
Positive	16	8	5	3	—
Negative	11	—	—	3	8

Positive Procaine Test—Pain and tenderness abolished; abduction restored temporarily.
Negative Procaine Test—Pain and tenderness abolished; abduction *not* restored.

Results—Analysis of the subsequent clinical course is shown in Table II. In six of the patients with negative procaine tests the shoulder cuff was explored later; all had tears involving the whole supraspinatus segment and in some the infraspinatus segment was also involved.

CONCLUSIONS

The series is small but the findings appear to justify the continued use of procaine infiltration as an aid to initial assessment of shoulder cuff tears. A positive procaine test indicates a fair prospect of satisfactory spontaneous recovery even in patients who show clinical evidence of an apparently severe lesion. On the other hand, a negative procaine test is an indication that spontaneous recovery is unlikely and that operative repair is advisable.

It is suggested that the early management of supraspinatus tears should be on the lines laid down in Table III.

TABLE III

Extent of tear	Treatment
CLINICALLY MILD	CONSERVATIVE
CLINICALLY SEVERE, but abduction temporarily restored by procaine infiltration	CONSERVATIVE in the first instance
CLINICALLY SEVERE and abduction not restored, even temporarily, by procaine infiltration	EARLY OPERATIVE REPAIR

SUMMARY

1. The end-results of conservative treatment of supraspinatus tears have been studied in a series of 109 patients graded on a clinical basis, and in a further series of twenty-seven patients assessed initially by procaine infiltration.

2. In 87 per cent. of patients with mild lesions, full function was regained in an average period of five and a half weeks. In more than 50 per cent. of patients with apparently severe lesions, there was full functional recovery in eleven to thirteen weeks.

3. Clinical assessment, other than as mild or apparently severe, is unreliable in the early stages.

4. Procaine infiltration of recent tears, by abolishing pain and spasm, allows more accurate assessment of supraspinatus function and gives a more clear indication as to the advisability of conservative or early operative treatment. If such infiltration of the torn segment of tendon fails to restore voluntary abduction power, early operative repair is indicated.

5. Six patients with negative procaine tests, in whom the shoulder cuff was subsequently explored, all showed extensive tears.

The author wishes to thank Mr Roland Barnes for generous help and valued criticism.

REFERENCES

BOSWORTH, D. M. (1941): The Supraspinatus Syndrome—Symptomatology, Pathology and Repair. Journal of the American Medical Association, **117,** 422.

CODMAN, E. A. (1934): The Shoulder. Rupture of the Supraspinatus Tendon and other Lesions in and about the Subacromial Bursa. Boston. Privately printed.

MCLAUGHLIN, HARRISON W. (1945): Lesions of the Musculo-tendinous Cuff of the Shoulder—Differential Diagnosis of Rupture. Journal of the American Medical Association, **128,** 563.

SHOULDER PAIN

With Particular Reference to the " Frozen " Shoulder

F. A. SIMMONDS, LONDON, ENGLAND

From St Thomas's Hospital and The Rowley Bristow Orthopaedic Hospital, Pyrford *

It is believed from investigation of patients with " frozen shoulder " that the syndrome can be correlated with certain other types of shoulder pain. It is characterised by a well-known cycle of events: 1) there is increasingly severe pain in the shoulder, spreading down the arm, sometimes developing after injury, in a patient aged fifty to sixty years; 2) the pain persists and gleno-humeral movement decreases until only about twenty degrees remain; 3) the pain becomes less severe but stiffness persists; 4) the pain subsides and movement is slowly regained. The cycle may take from six months to two years. It is usually believed that the prognosis is excellent and that there is nearly always complete recovery (Codman 1934, Wilson 1943, Ferguson 1938, Lippmann 1944, Moseley 1945); but this has not been our experience. In a small series of twenty-one patients who suffered from " frozen " shoulder more than three years ago only six regained normal function; nine have both weakness of the joint and persistent pain; and six complain either of weakness or loss of movement.

It is often stated that the movements that are lost are external rotation and abduction; but the fact is that there is approximately equal limitation of movement in all directions from the position in which the limb is rested. If the limb is supported by the side of the trunk in a sling, abduction is limited; but if it is splinted in abduction the shoulder " freezes " in that position.

Pathology—In four patients with " frozen " shoulder, parts of the bursa, supraspinatus tendon and biceps tendon were removed for histological examination and for comparison with similar specimens obtained at autopsy in subjects of the same age (Fig. 1). The fact that in these cases there was no increase in the range of movement when the patient was deeply anaesthetised indicated that the stiffness was not due to spasm. The subacromial bursa appeared thickened and oedematous and it showed increased vascularity, but there were no adhesions between its walls. The tendinous cuff also showed increased vascularity and it seemed abnormally thick and closely applied to the head. The cuff could be likened to a vascular, leathery hood with no obvious demarcation between the tendons. Attempted movement demonstrated that there were tight inelastic tissues all round the joint. The biceps tendon moved freely and was reddened only where it was in contact with the supraspinatus (contrast Lippmann, 1943); and excision of the tendon permitted no increase in the range of movement. The joint itself was normal and there were no intra-articular adhesions. *Microscopy* of the bursa and the tendon confirmed the naked-eye findings. The bursa showed chronic inflammatory reaction with hyperaemia. The supraspinatus tendon showed similar inflammatory changes spreading inwards from the periphery. There was evidence of degeneration and focal necrosis with marked increase of vascularity (Fig. 2).

DEGENERATIONS AND INFLAMMATION OF THE SUPRASPINATUS TENDON AND CAPSULE OF THE SHOULDER

Some authors have believed that the primary changes in " frozen " shoulder are in the subacromial bursa but it has been shown by Codman (1934) and other surgeons who have made special studies of the shoulder that these changes are merely secondary; and this is confirmed by our observations. Simple bursitis nearly always clears up rapidly with rest, and although chronic bursitis might cause persistent effusion and adhesions it is a very

* *Paper read at the Annual Meeting of the British Orthopaedic Association, October* 1948.

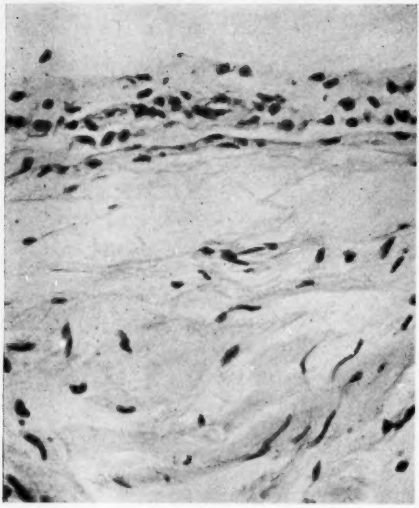


FIG. 1

Section of peripheral part of supraspinatus tendon in a cadaver (age 55 years) showing poor staining of the collagen fibres and loss of the normal wavy appearance. The picture is that of degeneration without inflammatory reaction (×90).

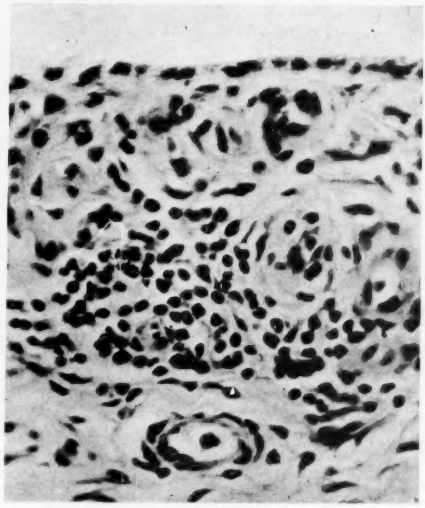


FIG. 2

Section of peripheral part of supraspinatus tendon in a male patient with "frozen" shoulder (age 57 years). The collagen is degenerate; several blood vessels with lymphocytes and histiocytes are seen; there is chronic inflammation (compare Fig. 1).

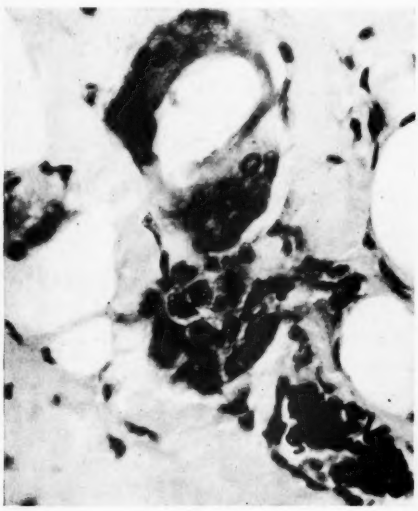


FIG. 3

High power view of section of supraspinatus tendon from same patient as shown in Figure 2 (×400). One huge multinucleate cell surrounds necrotic areas of tendon, some of which has dropped out of the section.

FIG. 4

Low power view of a section from a different part of the supraspinatus tendon in the same patient as shown in Figures 2 and 3 (haematoxylin and eosin, ×80) showing the extraordinary degree of vascularity.

uncommon lesion and no evidence of it was found in the cases of " frozen " shoulder that we explored at operation.

Biceps tendinitis and peritendinitis have sometimes been regarded as the cause of " frozen " shoulder but the tendon did not show significant changes in any of the shoulders we explored and it seems clear that biceps tendinitis is not a primary factor. The same is true of the joint itself. It must be assumed, therefore, that the basic cause of " frozen " shoulder does not lie in the joint, the biceps tendon, or the sub-acromial bursa. It is the fixator tendons and the capsule that are primarily affected. The whole tendinous cuff has an abnormal appearance but it is the supraspinatus tendon, which shows evidence of a vascular reaction around areas of degeneration, that is essentially involved. This is supported by certain observations. 1) " Frozen " shoulder seldom occurs when the supraspinatus tendon is torn and retracted; the typical clinical picture of such a lesion is that of painless loss of function with full passive movement (Codman 1934, Wilson 1943, Ferguson 1938). It is the incomplete tear, which is characteristically painful, that may proceed to the " frozen " shoulder, whereas in the complete tear that has been sutured a longstanding painful shoulder may develop. 2) Pathological changes occur in the supraspinatus tendon far more commonly than in the other tendons of the cuff. This is not surprising because the supraspinatus tendon is the longest, and most exposed to injury from friction against the acromion process or the coraco-acromial ligament. Rupture is more frequent in this than in other tendons and there is good evidence that a tendon seldom tears unless it is degenerated or has impairment of its blood supply (Wilson 1943, Lindblom 1939, Cronkite 1935, McMaster 1933). Similarly, calcified deposits, which probably represent areas of necrosis, occur most commonly in the supraspinatus tendon. In autopsies of aged persons a high incidence of degenerations and tears of the supraspinatus has been recorded (Codman 1934, Wilson 1943, Wilson and Duff 1943, and Herman Wahren 1942). The fact that these lesions have often been symptomless is probably accounted for by the nature of the surrounding tendon tissue which in old age may be incapable of a vascular reaction. If, then, the supraspinatus is the site of the primary lesion in " frozen " shoulder it would be expected that the fundamental fault is degenerative. But the tendon is not so degenerate that a vascular response is impossible, or there would be no pain, and that is why the syndrome occurs at the age of fifty or sixty years rather than later.

It is submitted that the " frozen " shoulder syndrome is caused by a chronic inflammatory reaction in the supraspinatus tendon due to atypical features in the processes of degeneration that occur constantly in this tendon with advancing age (Figs. 3 and 4). The sequence of events is: 1) the supraspinatus undergoes irregular and widespread degeneration with focal necrosis; 2) the tendon reacts by inflammation as it would to a foreign body; 3) the inflammatory reaction is painful, spreads to the bursa, and causes inefficiency in the function of the supraspinatus; 4) the painful area is constantly injured by impact against the acromion process and the coraco-clavicular ligament, and the limb is therefore rested in the sling position; 5) the capsule and cuff, because of a nearby inflammatory process coupled with lack of use, become inelastic with consequent loss of movement in all directions from the sling position. If necrotic areas in the tendon are absorbed, and the tendon is revascularised, an inflammatory reaction is no longer invoked and a painless stiff shoulder develops which slowly regains its movement (Table I).

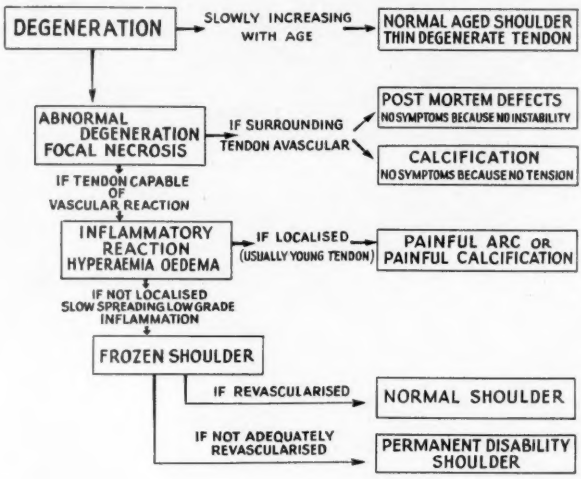
If the shoulder returns to normal, recurrence does not occur because the supraspinatus tendon has been revascularised; it may indeed have a better blood supply than before. But if the vascular response has been inadequate, tendon defects, tears of varying size, or secondary biceps tendinitis may develop. All stages between the weak but painless shoulder and the weak and painful shoulder may be seen.

The cause of the degenerative changes is difficult to determine, but it is probably associated with impairment of blood supply due to injury of the tendon by impact against

the acromion process and coraco-acromial ligament, especially in patients with cardio-vascular disease, and at the climacteric, when abnormal and irregular degeneration may be caused by sudden changes in vascularity. The "frozen" shoulder syndrome is more frequent when there is coincident cardio-vascular disease and, moreover, 60 to 65 per cent. of cases are in women (Lippmann 1944, Codman 1934). The average age of onset is a few years younger in women than in men (Codman 1934).

TABLE I

RESULTS OF DEGENERATION AND INFLAMMATION IN THE SUPRASPINATUS TENDON.


DEGENERATION
SLOWLY INCREASING WITH AGE
NORMAL AGED SHOULDER THIN DEGENERATE TENDON
ABNORMAL DEGENERATION FOCAL NECROSIS
IF SURROUNDING TENDON AVASCULAR
POST MORTEM DEFECTS NO SYMPTOMS BECAUSE NO INSTABILITY
CALCIFICATION NO SYMPTOMS BECAUSE NO TENSION
IF TENDON CAPABLE OF VASCULAR REACTION
INFLAMMATORY REACTION HYPERAEMIA OEDEMA
IF LOCALISED (USUALLY YOUNG TENDON)
PAINFUL ARC OR PAINFUL CALCIFICATION
IF NOT LOCALISED SLOW SPREADING LOW GRADE INFLAMMATION
FROZEN SHOULDER
IF REVASCULARISED
NORMAL SHOULDER
IF NOT ADEQUATELY REVASCULARISED
PERMANENT DISABILITY SHOULDER

Treatment—The aims of treatment of "frozen" shoulder due to degenerations and inflammations of the supraspinatus tendon and shoulder cuff should be: 1) to improve the blood supply; and 2) to preserve elasticity of the tissues. Increase in the blood supply may be promoted by radiant heat and diathermy, and by local interruption of the sympathetic nerves, but in practice these measures appear to be of little more than theoretical value. We have no experience of the hormone therapy that has been advocated (Walton, L. H. F. Personal communication).

Preservation of elasticity without inflicting further injury is not easy. Continuous traction which separates the tendon from its pressure points may be of value. Another method is to abduct the shoulder widely, restricting movement to an arc above the right angle, which was advocated by Codman. He rested the patient in bed and gradually pulled the limb into wide abduction. Once the great tuberosity had slipped under the acromion, patients were immediately more comfortable, and they began gleno-humeral movement in this highly abducted position. The chief difficulty is economic. Patients are unwilling to undergo prolonged hospital treatment unless the pain is very intense and a cure is guaranteed. As a rule it is necessary to accept the compromise of out-patient treatment with rest of the limb in a sling, gentle pendulum exercises, and perhaps novocain injection. All too frequently the sequel is a year or more of misery. Manipulation, or exercises in the erect position, are contra-indicated in the painful phase, because they lead to further injury.

BICEPS PERITENDINITIS

Primary biceps peritendinitis—The course of the long head of biceps through a narrow tunnel over a mobile joint renders it unusually liable to inflammatory changes at its surface and in the peritendon (Hitchcock and Bechtol 1948). Lippmann (1943, 1944) found evidence of biceps peritendinitis in all of thirty-two "frozen" shoulders on which he operated. He claimed that after fixation of the tendon in the groove there was rapid relief. It appears, however, from his clinical descriptions that few of these cases had the characteristic features of true "frozen" shoulder. In many of them the signs were those that we regard as typical of bicipital tendinitis—namely, pain in the shoulder and down the arm, tenderness over the bicipital groove, positive biceps tension test, and pain at the extremes of movement with or without a few degrees loss of movement. Our own view is that although biceps peritendinitis occurs as a primary lesion, causing shoulder pain which may simulate "frozen" shoulder, it is unusual for active or passive movements to be limited by more than a few degrees. Recovery from this type of painful shoulder might be hastened by anchoring the biceps tendon but the operation has no general application to all cases of "frozen" shoulder.

Secondary biceps peritendinitis—If there is a supraspinatus tear of sufficient size to impair stability of the humeral head the long head of the biceps acts as a fixator of the humeral head, though no doubt rather inefficiently. One of our patients who had signs of a supraspinatus tear found that he could abduct his arm only if he first increased biceps tension by full supination. At operation he had a large, recent, anterior supraspinatus tear; the biceps tendon, where it was exposed to injury by contact with the acromion, was inflamed. Other examples of biceps tendinitis associated with lesions of the supraspinatus have been encountered. Thus it seems that, when the long head of the biceps takes over the function of a fixator of the humeral head, the resulting strain and friction render it liable to secondary peritendinitis.

Treatment—*Primary biceps peritendinitis* usually recovers spontaneously if the limb is rested. In resistant cases, when the disability is severe, the tendon should be sutured to its groove, distal to the joint, the intra-articular portion being excised. Recurrent dislocation of the biceps tendon should be treated in the same way. *Secondary peritendinitis*—If secondary peritendinitis is due to a torn supraspinatus, and the tear is judged to be capable of healing after suture, the supraspinatus should be sutured and the biceps tendon excised, the stump being anchored in the groove. If the cause is an attrition defect, or a tear judged unsuitable for suture, the biceps tendon should be excised and its stump anchored; in these cases some loss of function must be expected. Lippmann (1943 and 1944) recommended fixation of the biceps tendon in the groove leaving the tendon in continuity. This shou'd give relief but not cure, because the useless intra-articular portion of the tendon buckles on movement and may act as a loose body. Excision of the intra-articular portion of the biceps, unless done blindly with a stripper, necessitates a small incision in the cuff. This cut should be made in the line of pull so that sutures are unnecessary and there is minimal danger of producing a painful shoulder from inflammatory reaction in the supraspinatus tendon.

THE PAINFUL ARC SYNDROME

The painful arc syndrome, which is sometimes associated also with constant aching, is occasionally due to bicipital peritendinitis or bursitis, but is usually due to local areas of degeneration in an otherwise healthy supraspinatus tendon, with low grade inflammatory reaction which causes pain. This local inflammation may be perpetuated by contusion of the tendon against the acromion. We have observed the late results in thirteen patients, the average age being thirty-eight years, although as a rule the syndrome occurs between the ages of forty and forty-five years. Three of five patients treated conservatively still have pain and weakness. Six of eight patients who were treated by excision of the acromion process have normal shoulders, the average pre-operative disability period being five years, and

the time necessary for full recovery after operation six months. One patient has a full range of movement and normal power after operation but complains of occasional pain; and the other unsatisfactory result of operation was in a patient who was greatly improved for nine months but then developed persistent signs of bicipital peritendinitis.

Calcification of the supraspinatus tendon—There are three clinical types of calcification of the supraspinatus tendon. In the first, the calcified deposit is entirely symptomless and is discovered incidentally. The second is characterised by aching pain in the shoulder and a painful arc of movement during abduction; there is localised inflammatory reaction but not enough tension to cause severe pain: these deposits remain in the substance of the tendon and as a rule they are slowly absorbed. In the third type there is very intense pain due to tension caused by the acute inflammatory reaction with muscle spasm. Often the tension is so great that rupture occurs either into the bursa or deep to it. When the tension is released, recovery is usually complete within a few weeks. Radiographs show the calcium deposit changing in position and shape and, as one would expect with a severe vascular reaction, the calcium disappears rapidly.

SUPRASPINATUS TEARS

In deciding whether or not to attempt suture of a ruptured supraspinatus tendon several points need to be considered. 1) The large tear, which causes instability of the humeral head, is rare; but, unless it is repaired, permanent disability is almost certain. 2) Retraction of the torn tendon increases, the longer suture is delayed. 3) Suture of a grossly degenerated tendon is unlikely to be successful. 4) Under experimental conditions rupture seldom if ever occurs in a tendon unless it is degenerated (Wilson 1943, Lindblom 1939, Cronkite 1935, McMaster 1933). In the living shoulder conditions are different, in so far as the supraspinatus tendon may be pinched between the humeral head and the acromion process at the same moment that the muscle is strongly contracted, so that tears may occur in young patients with only slightly degenerated tendons. 5) Suture of a supraspinatus tear may produce just those conditions which give rise to the " frozen " shoulder with a post-operative course that is painful and prolonged. 6) Secondary biceps tendinitis may be coincidental with defects in the supraspinatus tendon, perhaps especially when the groove in which the tendon lies is narrowed by suture.

The diagnosis of supraspinatus tears depends upon the fact that the supraspinatus is the chief fixator of the humeral head in abduction, the infraspinatus, subscapularis and biceps being relatively inefficient and capable only of stabilising the head against weak deltoid contraction. Provided pain has been relieved, the stability of fixation and power of abduction at the gleno-humeral joint is directly proportional to the amount of supraspinatus tendon still intact. Large tears with retraction are easy to diagnose because there is sudden onset of almost painless loss of function, often accompanied by a snap, with no restriction of passive movement but with total loss of the power of initiating abduction. More limited tears are difficult to differentiate from shoulder sprains; and small tears cause no appreciable weakness, pain being the dominant symptom. The differentiation is aided by noting the initial rhythm of abduction after procaine infiltration. If pain has been relieved, and the deltoid is acting strongly, disturbance of the initial rhythm (which normally is characterised by very little scapular movement during the first thirty degrees of abduction movement of the limb—and with no elevation of the shoulder) indicates instability of the humeral head and a supraspinatus tendon tear of considerable size.

Treatment of supraspinatus injuries—*Small tears* which are not the cause of instability cannot be distinguished clinically from sprains. Treatment should be by local heat, non-gravity exercises, and local injection of procaine. In this type of injury it may be impossible to prevent later development of the " frozen " shoulder syndrome. *Moderate tears* causing instability should usually be sutured. With or without suture, a " frozen " shoulder may

develop, but the ultimate prognosis ought to be better with suture. *Large complete tears* with retraction should be sutured if the expectation of life and capacity for work is good. In old patients who are unlikely to work again operation is inadvisable and the disability should be accepted.

Excision of the acromion process and biceps tendon in operations upon the supraspinatus—Routine excision of the intra-articular portion of the biceps tendon has two advantages: the possibility of later development of the bicipital syndrome is avoided; and the removal of an unnecessary and bulky structure from between the head of the humerus and the vulnerable area of the supraspinatus tendon reduces the likelihood of impingement against the acromion process and the coraco-acromial ligament. Our autopsy investigations indicate that this ligament is a strong and unyielding structure against which the anterior third of the supraspinatus insertion impinges when the efficiency of the supraspinatus is impaired. Impingement of the tendon against either the coraco-acromial ligament or the bone itself is relieved when the acromion process is removed as far proximally as the acromio-clavicular joint.

SUMMARY

1. The "frozen" shoulder syndrome is due to an inflammatory lesion in the musculo-tendinous cuff invoked by a local area of degeneration.

2. The available evidence suggests that the primary site of the degenerative lesion is in the supraspinatus tendon.

3. Other causes of shoulder pain which must be differentiated from "frozen" shoulder are peritendinitis of the long head of biceps, degeneration or tears of the supraspinatus, and calcified deposits in the supraspinatus.

4. An explanation of the pathogenesis of lesions of the musculo-tendinous cuff is submitted in which the different types of clinico-pathological syndrome are correlated. This hypothesis is in accord with the experimental, clinical and operative findings.

My thanks are due to Mr A. E. Clark of St Thomas's Hospital for his technical work in preparing the sections and photomicrographs. I am grateful to Professor W. G. Barnard for his opinions on the sections, and to Professor George Perkins for his stimulation and interest.

REFERENCES

CODMAN, E. A. (1934): The Shoulder. Boston, Massachusetts. (Privately printed.)

CRONKITE, A. E. (1935): The tensile strength of human tendons. Anatomical Record, **64,** 173.

FERGUSON, L. K. (1938): Five year review of shoulder pain due to lesions of the subdeltoid bursa and supraspinatus tendon. Surgery, Gynecology and Obstetrics (International Abstract of Surgery), **66,** 472.

HITCHCOCK, H. H., and BECHTOL, C. O. (1948): Painful shoulder. Observations on the rôle of the tendon of the long head of the biceps brachii in its causation. Journal of Bone and Joint Surgery, **30 A,** 263.

LINDBLOM, K. (1939): On pathogenesis of ruptures of the tendon aponeurosis of the shoulder joint. Acta Radiologica, **20,** 563.

LIPPMANN, R. K. (1943): Frozen shoulder; bicipital tenosynovitis. Archives of Surgery, **47,** 283.

LIPPMANN, R. K. (1944): Bicipital tenosynovitis. New York State Journal of Medicine, **44,** 2235.

MCMASTER, P. E. (1933): Tendon and muscle ruptures. Clinical and experimental studies on the causes and location of subcutaneous ruptures. Journal of Bone and Joint Surgery, **15,** 705.

MOSELEY, H. F. (1945): Shoulder lesions. Springfield: Charles C. Thomas.

WAHREN, HERMAN (1942): Subdeltoid bursa and shoulder joint. Acta Chirurgica Scandinavica, **87,** 353.

WALTON, L. H. F. (1948): Personal communication.

WILSON, C. L. (1943): Lesions of supraspinatus tendon. Archives of Surgery, **46,** 307.

WILSON, C. L. (1948): Experimental degeneration of the supraspinatus tendon. Journal of Bone and Joint Surgery, **30 A,** 769.

WILSON, C. L., and DUFF, G. L. (1943): Pathologic study of degeneration and rupture of the supraspinatus tendon. Archives of Surgery, **47,** 121.

PAINFUL SHOULDER

Calcification of the Supraspinatus Tendon

G. BLUNDELL JONES, EXETER, ENGLAND *

There is a particular type of painful shoulder that has been recognised as a clinical entity but in which the occurrence of spontaneous recovery has not been emphasised sufficiently. The onset of pain is sudden, very severe, and not usually precipitated by injury, often starting during simple movement of the arm or even by turning over in bed. All movement is limited by pain and muscle spasm and the limb is held rigidly to the side; there is inability to rest or sleep and some patients have become mentally strange after several days of such pain. Radiographs taken shortly after the onset of symptoms show calcification in the area of the supraspinatus tendon.

The condition is uncommon; it occurred in 1 per cent. of three hundred painful shoulder lesions reviewed at the Princess Elizabeth Orthopaedic Hospital, Exeter. Six cases are presented (Table I) in which the treatment consisted of simple rest with heavy sedation to assure sleep, followed by active movements within the limits of pain as symptoms subsided.

TABLE I

ANALYSIS OF SIX CASES OF CALCIFICATION OF THE SUPRASPINATUS TENDON, AND ONE OF CALCIFICATION IN TENDON NEAR THE HIP JOINT

Sex	Age	Occupation	Duration of symptoms	Calcification in first radiograph	Recovery of full movement	Full function	Disappearance of calcification in radiograph	Total duration of disability
M	30	Army Officer	7 days	Dense	14 days	21 days	21 days	28 days
F	40	Housewife	14 days	Dense	28 days	36 days	4 months	50 days
M	49	Clerk	2 days	Dense	36 days	42 days	3 months	46 days
M	30	Hotel Manager	2 days	Moderate	7 days	14 days	3 months	16 days
F	35	Housewife	2 days	Moderate	15 days	15 days	15 days	17 days
F	48	Housewife	6 days	Moderate	16 days	16 days	16 days	22 days
M	29	Army Officer	1 day	Dense (Hip joint)	15 days	28 days	28 days	28 days

Spontaneous recovery with absorption of the calcified deposit took place in fourteen to twenty-eight days and there has been no recurrence in a follow-up period of six months to five years. One case of a similar lesion of the hip joint is added because it presents the same clinical and radiographic features. Illustrative radiographs are shown in Figures 1–8.

* *Paper read at the Annual Meeting of the British Orthopaedic Association*, 1948.

FIG. 1

January 1, 1945

FIG. 2

January 20, 1945

Case 1. Male, aged 36 years. At the time of onset of acute pain and muscle spasm a dense opacity was shown in the radiograph (Fig. 1). Three weeks later, after simple rest, the radiographic appearances of calcification in the tendon had almost completely disappeared (Fig. 2).

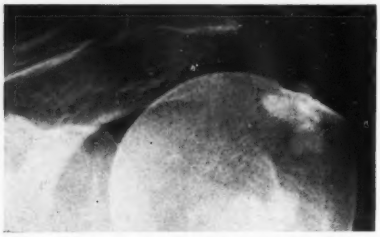


FIG. 3

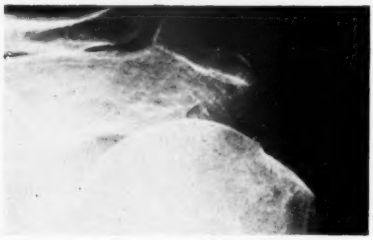


FIG. 4

Case 2. Female, aged 40 years. Figure 3 shows the condition fourteen days after the acute onset of symptoms. There was rapid improvement with rest. Radiograph four months later shows disappearance of the calcified deposit (Fig. 4).

FIG. 5

FIG. 6

Case 3. Male, aged 49 years. Figure 5 is the initial radiograph taken at the time of first onset of symptoms, showing an extensive calcified deposit. Within four weeks the opacity was of diminished intensity and after ten weeks absorption of the calcified deposit was almost complete (Fig. 6).

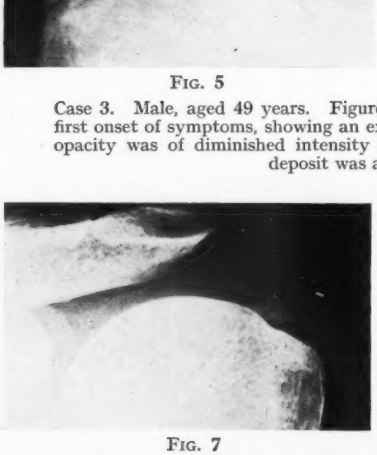


FIG. 7

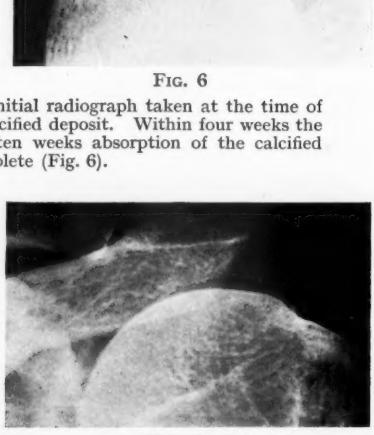


FIG. 8

Case 4. Male, aged 30 years. Figure 7 shows an opacity of moderate density. Three months later the radiographic appearances were normal (Fig. 8).

Discussion—There is a vast literature dealing with calcification around the shoulder joint. Codman (1934) gave a clear description of such deposits in the sub-deltoid bursa, and it has been recognised that this lesion is capable of spontaneous recovery. Nevertheless reports are still published which claim benefit for a particular treatment. Injection therapy is one such method. For example, Patterson and Darrach (1937) described a technique of removing the deposit by irrigation through two or more needles. Relief was obtained despite the fact that radiographs " sometimes show apparently the same amount of calcium after irrigation as before." Lapidus (1943), after reviewing the literature, presented a series of sixteen cases with acute symptoms together with sixteen cases in which other joints were affected. He found that treatment by infiltration with local anaesthetic and injection of saline into the calcified deposit caused relief of the symptoms and disappearance of the calcification soon afterwards. While recognising that the condition was capable of spontaneous recovery he concluded that injection therapy was beneficial. Recently, Norwich (1948) reported fifteen cases in which rapid recovery took place after injection therapy.

FIG. 9

FIG. 10

Case 7. Male, aged 29 years. Calcification in tendon near the hip joint shortly after the onset of symptoms (Fig. 9). The symptoms subsided gradually during rest in bed and four weeks later the area of opacity is greatly diminished (Fig. 10).

This writer believes that proper assessment of the natural repair processes in acute cases has not been made. Relief of pain can be obtained by simple rest and sedation without the added risk of infection by injected fluids. Objectively the recovery with such treatment has been as rapid and complete as that reported after injection therapy. Reactive hyperaemia after rupture of the deposit into the sub-deltoid bursa probably accelerates absorption and repair. Thus the more acute the symptoms, the more rapid the spontaneous cure.

SUMMARY

1. The explosive type of painful shoulder due to rupture of a calcified deposit into the sub-deltoid bursa is described.

2. A brief report of six cases is presented.

3. No treatment other than rest and sedation is needed.

REFERENCES

CODMAN, E. A. (1934): The Shoulder. Boston: Privately printed.
LAPIDUS, P. W. (1943): Surgery, Gynecology and Obstetrics, **76**, 715.
NORWICH, I. (1948): Surgery, Gynecology and Obstetrics, **86**, 183.
PATTERSON, R. L., and DARRACH, W. (1937): Journal of Bone and Joint Surgery, **19**, 993.

EXCISION OF THE ACROMION IN TREATMENT OF THE SUPRASPINATUS SYNDROME

Report of Ninety-five Excisions *

J. R. ARMSTRONG, LONDON, ENGLAND

The majority of patients who complain of a painful shoulder are suffering either from periarthritis, "frozen shoulder," or from a supraspinatus lesion. Lesions of the supraspinatus tendon at or close to its insertion, where it is intimately associated with the subacromial bursa, give rise to a characteristic combination of symptoms—the supraspinatus syndrome—which are mechanical in origin. In the middle range of abduction movement the tendon impinges on the overlying processes and the tendon and bursa are compressed between the humerus and acromion (Fig. 1). When there is abnormality of the tendon or bursa this pressure causes pain, and any movement which tends to bring the lesion into contact with the acromion causes reflex muscle spasm. The supraspinatus syndrome is usually characteristic, although in its later stages it may be complicated by true limitation of shoulder movement, due partly to disuse and partly to adhesion formation in the region of the subacromial bursa, which masks the typical symptoms and complicates both diagnosis and treatment.

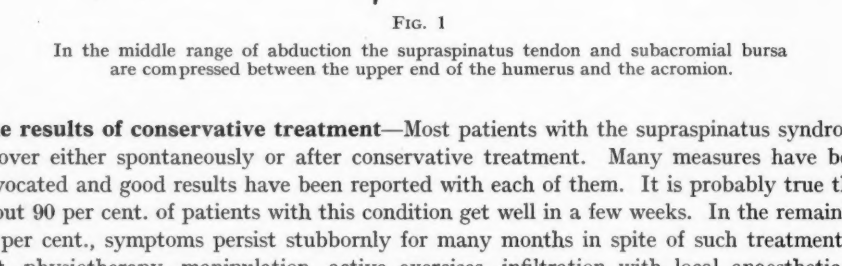


FIG. 1

In the middle range of abduction the supraspinatus tendon and subacromial bursa are compressed between the upper end of the humerus and the acromion.

The results of conservative treatment—Most patients with the supraspinatus syndrome recover either spontaneously or after conservative treatment. Many measures have been advocated and good results have been reported with each of them. It is probably true that about 90 per cent. of patients with this condition get well in a few weeks. In the remaining 10 per cent., symptoms persist stubbornly for many months in spite of such treatment as rest, physiotherapy, manipulation, active exercises, infiltration with local anaesthetic or deep X-ray therapy, and it is in this group of cases that excision of the acromion is indicated.

THE AIMS AND PRINCIPLES OF EXCISION OF THE ACROMION

The principle underlying excision of the acromion is simple. Intermittent pressure associated with abduction or forward flexion of the arm causes pain and muscle spasm, and constantly repeated irritation prevents healing of the lesion. Excision of the acromion relieves this pressure. A full range of painless shoulder movement then becomes possible and most of the symptoms disappear by the time the patient has recovered from the immediate effects of the operation. When relieved of irritation, the underlying condition slowly resolves or heals.

* *Based on a paper read at the Annual Meeting of the British Orthopaedic Association in Belfast, October* 194

To ensure successful results a considerable amount of bone must be removed. This point was not at first appreciated and in the first nine cases in this series not enough of the acromion was excised; in attempting to preserve the acromio-clavicular articulation the bone was divided immediately lateral to the joint (Fig. 3). At operation there appeared to be adequate clearance but the results were unsatisfactory in five of these patients. The upper end of the humerus lies slightly anterior to, rather than immediately below, the

FIG. 2

After excision of the acromion four and a half years previously the bone has reformed almost completely.

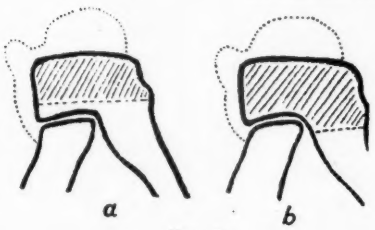


FIG. 3

Excision of bone distal to the acromio-clavicular joint (Fig. 3*a*) is not always satisfactory. It is better to remove the acromion completely (Fig. 3*b*) because otherwise new bone may form in the attachment of the deltoid to the raw bone surface.

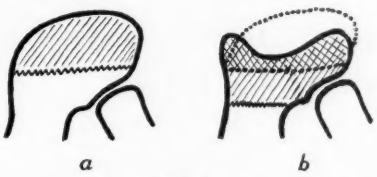


FIG. 4

Fig. 4*a* is a tracing of the line of first excision of the acromion in the case shown in Fig. 2. New bone that formed within a few months (Fig. 4*b*) necessitated a second operation for complete excision at the joint level.

acromion process and the insertion of the supraspinatus tendon comes into contact with the anterior part of the process on shoulder movement. In fact Codman suggested that it was contact with the coraco-acromial ligament rather than with bone that caused the pressure. In any event it is essential that the anterior part of the acromion should be removed completely and any attempt to preserve the acromio-clavicular joint may cause unsatisfactory results. Moreover, when the acromion has been divided and the detached deltoid muscle is sutured to its cut edge, new bone formation takes place rapidly and symptoms may recur (Figs. 2–4). If the whole of the acromion process lateral to the acromio-clavicular joint is

FIG. 5

FIG. 6

FIG. 7

Photograph of patient four months after complete excision of the right acromion (Fig. 5) and radiograph of the right shoulder (Fig. 6) and the left shoulder (Fig. 7). The difference in contour of the shoulder is scarcely noticeable.

removed, the end of the clavicle constitutes the new attachment to which the deltoid is sutured, so that no raw bone is exposed in the area in which new bone formation would be harmful.

Complete excision of the acromion does not appear to cause untoward effects. If the conoid and trapezoid ligaments are undamaged, loss of the acromio-clavicular joint is not associated with any disability. The cut edge of the deltoid muscle, sutured firmly to the coraco-acromial ligaments, forms a firm fibrous scar which gives support to the outer end of the clavicle. The operation does not result in any serious cosmetic blemish. The scar is of course visible and may tend to stretch a little, but the alteration of contour is not noticeable (Figs. 5 and 6). The posterior angle of the cut edge of bone should be rounded off so that no spike is left.

INDICATIONS FOR EXCISION OF THE ACROMION

Broadly speaking, excision of the acromion is indicated in treatment of the supraspinatus syndrome whenever conservative treatment has failed. More precise indications vary according to individual opinion and they depend upon a number of circumstances. The most important single factor is the duration of symptoms. Lesions of the supraspinatus group tend to resolve and subside with time, and operation is never a matter of urgency. Indeed some surgeons maintain that all these lesions recover spontaneously over a period of one or two years; but even if this were true, which is to be doubted, few patients are prepared to tolerate symptoms for so long a time, nor is it reasonable to expect them to do so. On the other hand it is impossible to tell in the early stages which patient will recover and which will require operation. Conservative treatment should always be tried for at least two months; and only if there is no improvement at the end of that time should operation be considered.

When, as is often the case, symptoms improve to some extent without being completely relieved, other factors must determine whether or not operation should be advised. The severity of the symptoms and the degree of associated disability are obviously important. In its initial stages the acute syndrome may be acutely painful and crippling, but this phase does not often persist. The typical syndrome is neither very painful nor completely incapacitating and its effects often depend on the age, occupation and mode of life of the patient. A painful arc of movement may be no more than a mild nuisance to an elderly patient of sedentary habits and yet be a severe handicap to a younger person whose occupations are strenuous. Discomfort, accepted philosophically by one with a phlegmatic temperament, may be devastating in its effects on a more highly strung patient. Each patient who does not respond to conservative treatment provides an individual problem; and the surgeon's attitude to operation will certainly be influenced profoundly by his opinion of its efficacy.

There are two circumstances in which excision of the acromion is contra-indicated. When there is true limitation of shoulder movement, operation produces a stiff and stubborn joint which requires months of treatment before mobility is restored. Muscle spasm can be distinguished from adhesion formation by examination after the lesion has been infiltrated with local anaesthetic or, better still, by examination under a general anaesthetic. If there is true limitation this must be dealt with by active exercises, and perhaps manipulation, before operation is contemplated. Operation is also contra-indicated if there is doubt as to the diagnosis. Removal of the acromion will relieve pressure on the supraspinatus tendon or subacromial bursa but if applied in a haphazard manner to the treatment of all stiff painful shoulders the operation gives very unsatisfactory results, particularly in patients with periarthritis.

RESULTS

In 1939, Watson-Jones first reported excision of the acromion in the treatment of supraspinatus tendon lesions at a meeting of the British Orthopaedic Association in Oswestry; and he described the procedure in 1943. At about that time this writer was becoming increasingly dissatisfied with the results of conservative treatment in many service patients who were under treatment at the Royal Air Force Hospital, Rauceby. Those who were not promptly relieved by conservative treatment were often incapacitated for long periods and there seemed nothing else to offer. After trial of excision of the acromion the results were encouraging, particularly when experience had shown that it was necessary to excise the whole of the acromion. Conviction that the operation was a good one was strengthened by personal experience. In 1944, after several months of typical incapacity which had persisted unchanged despite all forms of conservative treatment, my own acromion process was excised by Sir Reginald Watson-Jones with complete and permanent cure. The results in a personal series

of ninety-five patients are summarised in Table I. Because many of these patients were referred for operation after conservative treatment elsewhere had failed, it is not possible to state the proportion that they represent of all patients suffering from the supraspinatus syndrome.

In eighty of ninety-five patients the operation was completely successful; they were relieved of symptoms and regained a full range of powerful shoulder movement. In the first nine operations the acromion was divided far enough laterally to preserve the acromio-clavicular joint. Five of these patients gained incomplete relief or none at all. In three patients further excision of bone gave good results. Two re-excisions were carried out about two months after the original operation; the third patient gained initial relief from the first operation but symptoms then recurred and at the time of re-excision seven months later new bone formation was obvious. In the other two unsuccessful cases permission for further operation was refused; one patient was satisfied with the incomplete relief he had gained; the other was discouraged by the unchanged persistence of his original symptoms. In six instances the result of operation was unsatisfactory because there was post-operative

TABLE I
RESULTS OF OPERATION

RESULTS OF EXCISION OF THE ACROMION IN NINETY-FIVE PATIENTS SUFFERING FROM THE SUPRASPINATUS GROUP OF LESIONS	
Satisfactory to patient and surgeon	80 (84·2 per cent.)
Unsatisfactory	15 (15·8 per cent.)
ANALYSIS OF FIFTEEN UNSATISFACTORY RESULTS	
Insufficient bone removed	5 (In three patients a further excision was successful)
Post-operative limitation of movement	6
No relief of symptoms	4

limitation of shoulder movement; there was complete relief of pain but the range of forward flexion and abduction movement was reduced by about one-third and the patients, being content with relief of pain, were unwilling to be stimulated in making the endeavour needed to regain a normal range of movement. Four patients said that they had gained no relief at all: in two the original diagnosis may have been inaccurate; and in the other two failure of the operation is still unexplained.

PATHOLOGICAL FINDINGS AT OPERATION

The subacromial bursa was opened at operation in every case and the pathological findings were noted. The various conditions that were seen were not always distinctively different, one from the other. Nevertheless it seemed possible to classify them into four groups (Table II).

Fifty-six patients appeared to have tendinitis with an associated bursitis. The tendon was red, thickened and rough; and the bursal walls and synovial lining were oedematous

and inflamed, these changes being most marked around the tendon. In recent and acute cases the synovial membrane hung in red, oedematous folds. The changes suggested that tendinitis was the primary lesion and bursitis secondary.

In ten patients the primary lesion appeared to be subacromial bursitis. The walls of the bursa were red, thickened and adherent to the acromion; the cavity contained free fluid and, in a few instances, small loose bodies. The changes were uniform, and not in any way localised to the region of the supraspinatus tendon, and they were exactly similar to those of a chronically inflamed prepatellar or olecranon bursa.

In fifteen cases calcified deposits in the supraspinatus tendon were visible in the pre-operative radiographs. At operation the tendon appeared rough, thickened, opaque and slightly red, and there was evidence of localised bursitis. No attempt was made to remove the deposits from the tendon.

Tears of the supraspinatus tendon were found in fourteen patients. Most frequently the tendon was incompletely detached from its insertion. The area of detachment was small and not associated with retraction such as occurs after complete rupture. Occasionally small rents were observed in the tendon itself through which the articular cartilage of the humeral head could be seen.

TABLE II

PATHOLOGICAL FINDINGS

PATHOLOGICAL FINDINGS IN NINETY-FIVE PATIENTS WITH THE SUPRASPINATUS SYNDROME	
Tendinitis with secondary subacromial bursitis	56
Primary subacromial bursitis	10
Calcification in supraspinatus tendon	15
Tears or detachments of supraspinatus tendon	14

OPERATIVE TECHNIQUE AND POST-OPERATIVE MANAGEMENT

Certain points in the technique, which make the operation easier, are worthy of note. The patient is laid on the sound side with the head well flexed. The surgeon sits at the top of the table, the arm being controlled by an assistant. The incision begins in front of the acromio-clavicular joint and extends back across the joint and acromion in a direction slightly concave outwards. A flap of skin and subcutaneous tissue is then raised, exposing the upper surface of the acromion and joint. The periosteum is divided about half an inch lateral to the proposed line of section and reflected inward. The acromion is divided from before backwards with a sharp osteotome held very obliquely to avoid the possibility of damage to underlying structures. The line of section should extend directly backwards from the acromio-clavicular joint. When the acromion has been divided it is held in lion forceps while the deltoid origin is detached from its outer edge, working from behind forwards. The subacromial bursa is usually found to be adherent and must be dissected from the deep surface of the bone. The last structures to be divided are the acromio-clavicular and coraco-acromial ligaments.

After removal of the acromion the bursa is opened for examination, and the underlying tendon is inspected, the arm being abducted and rotated as necessary. The bursal wall is closed. The cut edge of the deltoid is sutured firmly to the acromio-clavicular ligaments and reflected periosteum or, if necessary, to the bone itself through holes drilled with an awl. The deltoid is repaired while the limb is held in abduction and security of the suture-line is then tested by lowering the arm to the side. The skin is sutured and a pressure bandage applied.

During operation some generalised arteriolar bleeding occurs, but no large vessels are encountered and few ligatures are necessary. It is very much easier and quicker to remove the acromion in this retrograde manner than to attempt to clear its muscle attachments and then to divide the bone. The acromion should always be cut cleanly with a sharp osteotome and never be nibbled away piecemeal.

Post-operative management—After operation it is unnecessary to immobilise the limb in abduction. The patient wears a sling but is encouraged to use the forearm and hand as much as possible. Passive movements of the shoulder are permitted, the arm being placed in the most comfortable position or rested on a pillow. The patient need be confined to bed only for four or five days. Active movements of the shoulder are not attempted for ten days after operation during which time contraction of the deltoid is painful; but after this interval gentle shoulder exercises should be encouraged, particular attention being paid to abduction. In the early stages exercises are best tolerated in the supine position which minimises the effects of gravity, and it is advisable to continue abduction exercises in this position until a full range has been regained.

There is of course much variation in the rate of recovery of different patients. An active man may play golf without difficulty within four weeks of operation; but most patients need six to eight weeks before they are able to use the shoulder with confidence. The last ten degrees of abduction and forward flexion movement are regained slowly, and very often, since the patient may not appreciate that there is still some limitation of movement, it is difficult to persuade him to persist assiduously with the necessary exercises.

Acute symptoms are at once relieved by the operation but often there is slight aching, especially at night, for several months. The cure may be regarded as complete when the patient has not only regained a full range of movement but is also able to sleep comfortably at night on the affected limb.

CONCLUSIONS

1. The supraspinatus group of lesions constitutes one of the two common causes of the painful shoulder.

2. Most, but not all, of these lesions resolve either spontaneously or after conservative treatment.

3. When conservative treatment fails symptoms can be relieved by excision of the acromion process, provided that sufficient bone is removed to relieve all pressure on the tendon throughout a full range of shoulder movement.

4. Excision of the acromion is contra-indicated if there is doubt as to the diagnosis or if there is true limitation of shoulder movement.

REFERENCES

WATSON-JONES, R. (1939): "Excision of acromion for supraspinatus tendinitis." Demonstration of cases at meeting of British Orthopaedic Association, Oswestry.

WATSON-JONES, R. (1943): Fractures and Joint Injuries. Third edition. 418. Edinburgh: E. & S. Livingstone, Ltd.

VITAMIN E THERAPY IN DUPUYTREN'S CONTRACTURE

Examination of the Claim that Vitamin Therapy is Successful

RAYMOND A. KING, LONDON, ENGLAND

From the Orthopaedic and Accident Department, The London Hospital

It has been claimed that striking success can be gained in the treatment of Dupuytren's contracture of the palmar fascia by simple oral administration of vitamin E in high dosage. It has been said that, after such treatment, thickening of the fascia disappears and contracture of the fingers is relieved. Le Roy Steinberg (1947) used daily doses of 300 mg. of mixed natural tocopherols (of which 60 per cent. is in the form of alpha-tocopherol) for a few weeks until maximal effect was gained. A daily maintenance dose of 1 mg. per kilo of body weight was continued thereafter.

Such a claim of success from so simple a treatment warranted careful investigation and a trial series of thirteen patients was studied in the Orthopaedic and Accident Department of the London Hospital. The routine dosage was 100 mg. of pure synthetic alpha-tocopherol, three times a day, given in the form of Roche " Ephynal Forte " 20 mg. tablets. The aim was to continue treatment for about three weeks, or " until maximal improvement had been gained," and thereafter to give a smaller maintenance dose. In seven patients we succeeded in giving heavy doses for periods ranging from four to eight weeks; in four the treatment was abandoned during the third week; and in two it was abandoned during the first few days.

Four patients complained of headache, nausea, fatigue, drowsiness, " singing-in-the-ears," " swelling of the tongue," giddiness, blurred vision, sweating, and other symptoms that made it impossible to continue. The other nine patients made no such complaints, and four of them were quite sure that they had gained improvement. One, with a moderate degree of contracture, said: " the band feels softer." Another, with deformity of moderate degree, said: " the fingers feel straighter and the aching pain has gone." Two others had severe deformity and both insisted that " there is improvement " but were unable to specify the nature of the improvement.

The effects were checked in every case by detailed clinical records, accurate measurement of deformity, and serial clinical photographs. In twelve of the thirteen patients there was no evidence whatever of any alteration. In one, with moderate deformity, we think that after the second week of treatment the finger was somewhat straighter and the palmar thickening somewhat softer. Review one year later (after five weeks of full dosage and three weeks of maintenance dosage) shows that he still has moderate deformity. In the other twelve, including those treated on full dosage for six to eight weeks (at a cost of £5 a week), we can see no change. The treatment has been abandoned.

REFERENCE

STEINBERG, C. LE ROY (1947): " Fibrositis, including Dupuytren's Contracture—A New Method of Treatment." New York State Journal of Medicine, **47**, 1679.

THE STRENGTH AND ELASTICITY OF BONE IN RATS ON A RACHITOGENIC DIET

J. B. DE V. WEIR (Glasgow), G. H. BELL (Dundee) and
J. W. CHAMBERS (Glasgow), SCOTLAND

From the Institute of Physiology, University of Glasgow; the Department of Physiology, University College, Dundee, University of St Andrews; and the Department of Materia Medica, University of Glasgow

Since bone is primarily a supporting and protecting tissue it seems appropriate to investigate its properties in the same way that an engineer measures the characteristics of his particular structural materials, such as iron and steel. The orthopaedic surgeon, in dealing with bone, labours under certain disadvantages as compared with the engineer—he cannot ever gain complete control over living structures: yet at some time a knowledge of the strength and elasticity of bone is likely to be useful as well as interesting. The occurrence of fractures and other sudden changes is probably to be explained on the basis of static properties such as those measured by the engineer. Slower changes such as alterations in shape due to age or disease cannot be explained on this basis because bone, unlike iron, is a living material. The dynamic reactions of living bone have not yet been investigated quantitatively because suitable methods have not been found. In the meantime we must confine ourselves to static tests. The results of some of these tests in various conditions have already been presented (Bell, Cuthbertson and Orr 1941, Bell and Cuthbertson 1943, Bell, Chambers and Dawson 1947). In this paper it is proposed to deal almost entirely with the elastic properties of bone. Very little work has been published on this subject. We ourselves have made a few measurements on sheep bones (Bell and Weir 1949) and a few observations are recorded in Tabulae Biologicae (1925). The bones used for the work described in this paper were obtained in the course of experiments described by Bell, Chambers and Dawson (1947). Full details are provided in that paper but a summary of the experiments will be given here.

METHODS

Animals and diet—Male albino rats, after weaning at three weeks, were kept on a full diet until they reached roughly 50 g. in weight. The rats were then divided into three groups. Group R were put on a rachitogenic diet—the Steenbock diet No. 2965 (Steenbock and Black 1925); group N were given this rachitogenic diet with a supplement of vitamin D; group S were given a complete diet—Rowett Institute Stock diet (Thomson 1936). Litter mates were distributed among the three groups. The animals were killed after a period of about one month, the actual period of feeding varying from twenty-seven to thirty-six days. The femora were dissected out, cleaned carefully, and allowed to dry under ordinary room conditions before being subjected to the bending tests described below.

Bending tests—The method of carrying out a bending test, and the reasons for choosing it, are given by Bell, Cuthbertson and Orr (1941). It reproduces experimentally one of the strains to which living bones may be subjected by loads or muscular action. Moreover the bending test is a convenient method of applying a large stress (force per unit area) which at the same time gives an easily observable deformation (strain). Engineers have found it to be one of the most reliable tests of strength of a material.

The method of carrying out the test is indicated in Figure 1. The femur is laid on two supports C, C′. It is loaded at the middle by a hook D and pan carrying weights W. Semi-cylindrical end-pieces A, A′ of a hard resinous substance, cast on the ends of the bone, allow an accurate measurement of the span l, and prevent axial rotation. The load W is increased gradually until the bone breaks. As the bone is loaded the centre point of the shaft sags and the deflexion y is measured on a dial gauge DG resting lightly on the hook. The external breadth B, and depth D, of the shaft at the mid-point of the bone (Fig. 2) are taken before the test. The corresponding internal dimensions b and d are obtained by subtracting the thickness of the bone-wall as measured at the fracture site.

The bending moment M, which measures the bending force on the bone as a whole, depends on the span as well as the load. At the centre of the span it is given by $M=\frac{Wl}{4}$. When the bone is loaded the upper surface is compressed and becomes concave, and the " fibres " of the lower surface are in tension and become stretched. For any given load the stress, or force per unit area on the bone substance, is greatest in the outer " fibres " at the centre of the shaft where the load is applied. It diminishes towards the ends. This value is given by:—

$$\text{Stress} = \frac{MD}{2I} \text{ or } \frac{WlD}{8I} \quad \ldots\ldots\ldots (1)$$

where I is the moment of inertia of the cross-section of the bone at the site of fracture. Since the cross-section is nearly elliptical, I is given by

$$I = \frac{\pi}{64}(BD^3 - bd^3) \quad \ldots\ldots\ldots\ldots (2)$$

As the load is increased, the stress at the centre increases until it reaches a maximum value at which the bone breaks. This maximum value is called the breaking stress on bending, and it is denoted by S_B.

The change in length per unit-length is called strain. Like the stress it is greatest in the outer " fibres " at the mid-point of the span, where it is given by

$$\text{Strain} = \frac{6yD}{l^2\left\{1 - 8\left(\frac{c}{l}\right)^3\right\}} \quad \ldots\ldots\ldots (3)$$

The stiffness, or resistance to deflexion, of a bone depends on the size and shape of the cross-section and on the elastic quality of the bone substance. Young's modulus of elasticity, denoted by E, is a measure of the stiffness of the bone material. The stiffness of the bone as a whole is proportional to EI, and the flexibility or ease of bending is proportional to $\frac{1}{EI}$. The accepted formula for E, for a beam loaded at the centre, is $E = \frac{Wl^3}{48Iy}$ where I is the moment of inertia of the cross-section of the beam and y is the deflexion or sag at the centre. This formula is applicable only if the cross-section of the beam between the supports is constant but it can be applied, with slight modification, to the rat femur which is in fact somewhat thicker towards the ends. If a length c, at each end of the bone, is so supported by the semi-cylindrical end-pieces, or is so stiff as not to be bent

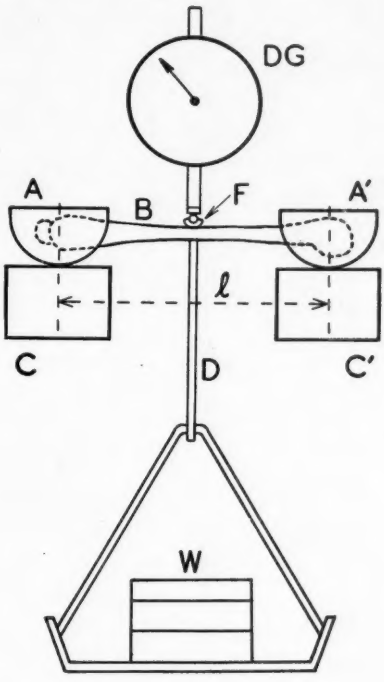


FIG. 1

Apparatus for measuring elasticity and bending strength of bones. B, bone. A, A', semi-cylindrical ends cast on to bone ends. C, C', supports. D, hook covered by layer of fibre F. W, load. DG, dial gauge. l, span.

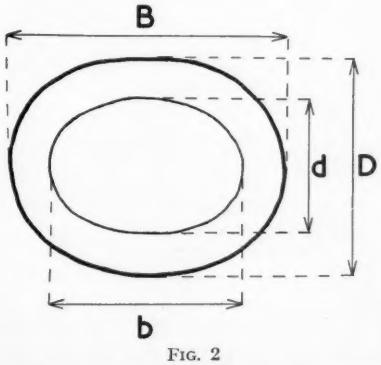


FIG. 2

Cross section of rat femur at the mid-point of the shaft to indicate the measurements taken.

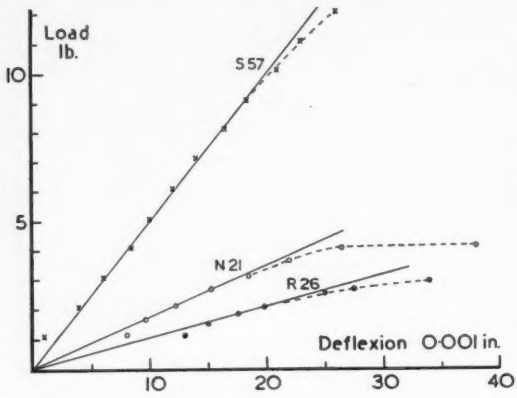


FIG. 3

Load-deflexion diagrams for three typical bones, one from each group. Group R = •, group N = o, group S = x.

appreciably when the bone is loaded, then a small "end-correction" can be made and the formula becomes:—

$$E = \frac{Wl^3}{48Iy}\left\{1 - 8\left(\frac{c}{l}\right)^3\right\} \quad \ldots . (4)$$

Even in an extreme case, where c is $\frac{l}{4}$ and all the bending takes place in the middle half of the shaft of the bone, the correction is only 12·5 per cent. Thus, when correction for the part of the bone embedded in the semi-cylindrical end-pieces is made, there can be only a small residual error due to increase in the size of the shaft towards the ends.

Chemical methods—After completion of the mechanical tests some of the femoral fragments were retained for X-ray crystallography. The rest of the bone shaft was ashed in a crucible at full Bunsen heat. Estimations of ash, Ca and P, were made as already described (Bell *et al.* 1947).

RESULTS

Rats on the rachitogenic diet without supplement, group R, all showed gross evidence of rickets as judged radiographically by widening of the upper tibial epiphysis. In group N, which received vitamin D in addition to the rachitogenic diet, the epiphysial lines were quite narrow. In group S, on a good diet, the femora were nearly twice as heavy as those in group N. Group R bones were about 20 per cent. lighter than group N bones. The average percentage ash content of the femora is shown in Table I. The individual results are plotted in Figures 5 and 6. As diet improved the ash content of the bones increased. The average length of group R femora was 2·0 cm.; of group N, 2·4 cm.; and of group S, 2·7 cm. The scatter of results tended to be greater in group R, because the small size of the bones made measurements more difficult.

The load-deflexion diagrams for three representative bones, one from each group, are given in Figure 3. The curve for group S bone is very nearly a straight line, except for the last quarter when the deflexion increases rather more rapidly than the load. In the N and R specimens there is greater deviation from a straight line. Over the straight part of each curve the deflexion is directly proportional to the load; the gradient of the curve indicates the stiffness (or resistance to deflexion) of the bone as a whole. Since the bones are of different sizes these graphs cannot be compared directly and they give no indication

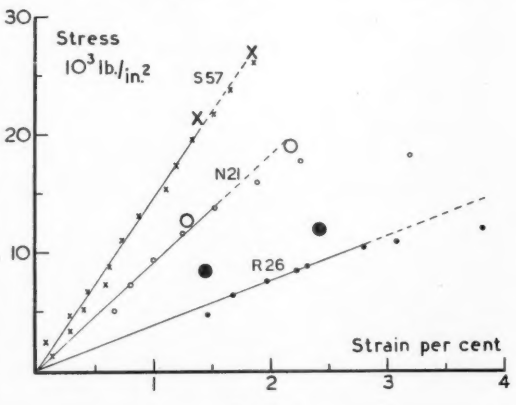


FIG. 4

Stress-strain diagram of the three bones of which the load-deflexion curves are given in Fig. 3. Group R = •, group N = o, group S = x. The large symbols are placed at the average values for each group of the stress and strain at the elastic limit and at the point of rupture.

of the quality of the material from which these bones are built. Direct comparisons of the bone material can however be made from stress-strain diagrams. Stress in the outer fibres is given by formula 1, and the strain by formula 3. These values have been calculated and are plotted in the stress-strain diagram Figure 4. The tangent of the angle made by the straight part of each curve (Fig. 4), and the x axis, is E or Young's modulus. The point at which the gradient begins to diminish indicates the upper limit of elasticity, beyond which plastic deformation occurs and ends in rupture. The breaking point gives the stress and strain at which the bone begins to break.

TABLE I

CHEMICAL AND PHYSICAL PROPERTIES OF BONES FROM RATS

R on a rachitogenic diet; N on a rachitogenic diet supplemented with vitamin D; and S on a complete diet

Group	No. of animals	Average ash per cent.	Average S_B 10^3 lb./in.^{2}	Average E 10^6 lb./in.^{2}	Average S_B / Average E $\times 10^2$	Average $S_B/E \times 10^2$	S.D. of $S_B/E \times 10^2$
R	12	37	11·9	0·64	1·85	2·04	0·65
N	19	43	18·9	0·99	1·90	1·91	0·20
S	6	59	26·9	1·56	1·73	1·74	0·11

TABLE II

PHYSICAL DATA FOR RAT BONES AT ELASTIC LIMIT AND AT RUPTURE

Group	R	N	S
Mean strain at elastic limit per cent.	1·45	1·28	1·39
Mean strain at rupture per cent.	2·41	2·18	1·84
Mean strain at elastic limit as percentage of mean strain at rupture	60	59	75
Mean stress at elastic limit 10^3 lb./in.^{2}	8·4	12·6	21·2
Mean stress at rupture 10^3 lb./in.^{2}	11·9	18·9	26·9
Mean stress at elastic limit as percentage of mean stress at rupture	70	67	79

While stress at the elastic limit is greater in bones with the higher ash content, strain is very much the same in all bones (Table II and Fig. 4). At breaking point, the stress is again greater the higher the ash content; but there is a tendency for the strain at rupture to be higher in bones produced on the poorer diets, when the percentage of ash is low. The differences which diet produces in E, S_B, and percentage ash, between the different groups of rats, are highly significant ($P<0{\cdot}001$—Student's t test).

In order to study the relationship between breaking stress, elasticity, and chemical composition of the bone material, we have made three diagrams (Figure 5, 6, 7). Figure 5, already given by Bell, Chambers and Dawson (1947), illustrates the relationship between the breaking stress and the percentage of ash in the bones. The diagram suggests that there is an association between breaking stress and percentage ash. These are intrinsic qualities of bone material, and they are independent of the actual sizes of the individual bones. The scatter diagram relating Young's modulus to percentage ash is very similar (Fig. 6).

The relation between S_B and E was also investigated (Fig. 7). A significant correlation was found within group N and group S taken separately. The combined correlation coefficient for the three groups R, N, and S, even after elimination of the effect of diet, is +0·63, which is still highly significant. Elasticity and breaking stress appear to be related more closely to each other than to the percentage of ash in the bone; but this is at least partly due to elimination of a source of variation common to both S_B and E, namely experimental errors in determining the moments of inertia of the very small cross-sections of the bones. Since the value of E is closely and positively correlated with the value of S_B, it is not surprising to find that there is no significant difference in the values of S_B/E in the three groups (Table I). The standard deviation of S_B/E is shown in Table I to give an idea of the scatter.

To test the possibility that rupture of the bone depends on deformation rather than load, we have attempted to estimate the ultimate strain when the bone was beginning to break. The formula for the strain, formula 3, is accurate only to the elastic limit of the material. Although the values obtained beyond this point cannot be regarded as absolute values, it seems justifiable to use them for comparative purposes. These values, together with the corresponding stress values, are given in Table II.

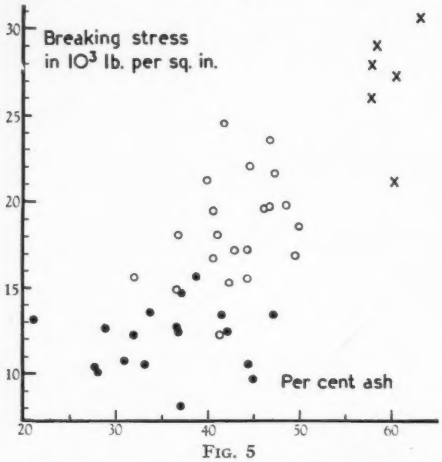


FIG. 5

This diagram shows the breaking stress S_B plotted against percentage ash in the bone. Group R = •, group N = o, group S = x. Each point refers to one bone.

CONCLUSIONS

One of the aims of this work was to find criteria by which the quality of bone as a supporting tissue might be judged. This inevitably involves discussion and, if possible, assessment, of the relative importance of the inorganic and organic material of the bone. It is relatively easy to measure the mineral content, and for that reason it has always received more than its due share of attention.

In the present experiment the composition of the ash of all bones was remarkably constant, with a Ca/P ratio of 2. Furthermore, X-ray crystallography showed that the structure of the inorganic material was the same in all cases. The great difficulty of measuring variations in the quality of the organic material which is, of course, protein in nature makes it impossible to say how much it influences bone strength. Since at least 40 per cent. of the bone is collagen, either a quantitative or a qualitative alteration might alter bone strength. X-ray crystallography revealed no qualitative differences in the collagen material of bones of the three groups; so that for the present it would seem safer to assume that alterations in the physical properties of the bones are due to variations in the relative proportions of organic and inorganic constituents (Dawson 1946, Bell *et al.* 1947).

These experiments show that the three diets produce highly significant differences in the percentage of ash, in S_B, and in E. It is possible that some variations in the percentage of ash are due to variations in the absolute collagen (weight of collagen in unit volume of bone substance); but the range of variation in the percentage of ash leaves no reasonable doubt that differences in percentage ash between the diet groups are due essentially to differences in absolute ash. Presumably the collagen contributes something to the strength of the bone; but the indications are that it plays a minor part and that the relative weakness and flexibility of rachitic bones is due to decrease in the absolute ash content. Within any

one diet group, the relation between percentage ash and the other two variables, S_B and E, is masked by other sources of variation such as those associated with the many measurements involved; and thus the correlation between percentage ash and S_B, and also between percentage ash and E, is not significant.

At first sight, the scatter diagrams (Figs. 5 and 6) appear to indicate a correlation between ash and S_B, and between ash and E. Closer inspection shows, however, that the apparent trend is due largely to differences between the means of the diet groups, and that the points within any one group show no such obvious trend. Figure 7 shows that the position with regard to correlation between S_B and E is very different. Here there is an obvious trend within each diet group; the amount of scatter is very much less. Calculation shows that, even when the differences between the means of diet groups is excluded, there is still a significant correlation between S_B and E. The question of the correlation between the three variables is discussed more fully in the addendum to this paper.

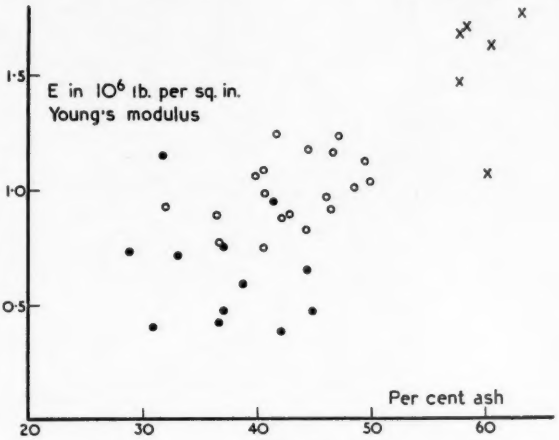


FIG. 6

This diagram shows Young's modulus, E, plotted against percentage ash in the bone. Group R = •, group N = o, group S = x. Each point refers to one bone.

Although the "goodness" of a bone is usually judged by its breaking stress, the experimental findings recorded above suggest that it may be assessed equally well on the basis of elastic properties as shown by Young's modulus. Normal bones, group S in these experiments, were elastic up to 79 per cent. of their breaking stress (Table II): the poorer bones of groups R and N were, however, only a little inferior in this respect. In some cases there was no apparent deviation of the load-deflexion curve from a straight line until the bone was about to break. Such a curve was published in the first paper of this series (Bell, Cuthbertson and Orr 1941), but in the light of further experience this curve is scarcely typical. The terminal falling over of the curve is illustrated in Figure 4 and is much more marked in the bones of group R.

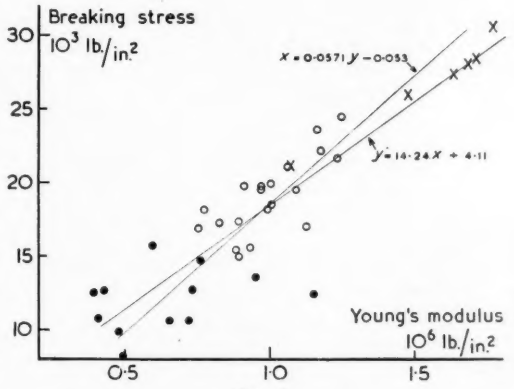


FIG. 7

This diagram shows the relationship between breaking stress S_B and Young's modulus E. Group R = •, group N = o, group S = x.

While stress at the upper limit of elasticity varies over a wide range in the three groups (Table II and Fig. 4), the strain at this point is remarkably constant at about 1·5 per cent. This same percentage displacement must occur between the molecules of the bone material at

the elastic limit—and it may be that, up to this amount of molecular displacement, the deformation is reversible; but that beyond it, plastic changes occur. We have no evidence as to whether the limiting displacement concerns mineral or protein constituents of the bone, or both.

We have already commented on the remarkable strength of bone material (Bell *et al.*, 1941). The breaking stress of normal rat bone is about the same as that of cast iron, and about half that of mild steel. Young's modulus, however, is only one-tenth that of cast iron and one-twentieth that of steel. Thus bone, despite its lightness (specific gravity about 2·5 as compared with 7·9 for iron), is remarkably strong and at the same time more flexible than might be expected. Presumably the biological advantage is that greater flexibility helps to absorb sudden impacts. It is unusual in metallic substances to find the elastic modulus proportional to the strength; this is more characteristic of materials like concrete and timber. Another remarkable property of bone is that it remains elastic up to three-quarters of the breaking stress. Most metals show considerable ductility before reaching their breaking point.

While Young's modulus is of interest, both on its own account and as an index of the quality of the bone, its close association with breaking stress suggests that it might be used to predict the maximum load which a bone can carry safely. Since E, unlike S_B, can be measured without damage, useful information might be gained by measuring the elasticity of living human bones.

ADDENDUM

WALTER L. M. PERRY, LONDON, ENGLAND

From the National Institute for Medical Research, Hampstead, London

During analysis of the data presented in this paper an interesting observation was made of the effect of the combination of groups of results on the overall correlation coefficient. If the three groups of observation—R, N and S—are considered, it is found that there is no significant correlation between ash and S_B, or between ash and E, in any one separate group. If the groups were simple replicates of the same experiment, with all variables other than the two under investigation controlled, then combination of the data would be legitimate, and the overall coefficient might reach a significant level owing to the larger number of observations concerned. In this investigation, however, the three groups are not simple replicates of the same experiment but are in fact three different dietary groups. Thus an additional variable, namely diet, has been introduced between the groups.

It might be argued that diet has an effect upon only one of the variables concerned—for example ash—and does not change any other property of the bone. This, however, is not known for certain. The diet might, for example, affect the quality of collagen of the bones as well as their ash content. In this way elasticity and breaking stress could vary independently of the ash content. In the absence of any information on this question it seems highly undesirable to combine the data from the three groups without eliminating the effect of differences in diet.

Statistical calculations—The total number of pairs of observations is, in each case, thirty-seven. A combined correlation coefficient may be calculated from these figures which is appropriate to the thirty-six degrees of freedom between them. It will be seen, however, that of these thirty-six degrees of freedom, two are attributable to differences between the means of the dietary groups, and only the remaining thirty-four to total correlation when these differences are eliminated. The total correlation on thirty-four degrees of freedom is readily obtained by summating the sums of squares used for the calculation of the correlation coefficients for each group separately, the degrees of freedom associated being eighteen in group N, five in group S, and eleven in group R. This gives a total as before of thirty-four. The results of these calculations are summarized in Table III. It will be seen that there is always a highly significant correlation when the effect of diet is not eliminated, but that only S_B and E are significantly related when this correction is made.

Conclusion—These results are of general interest in that combination of data such as these may often be carried out without appropriate corrections and thus high and misleading values may be obtained for the correlation coefficient.

TABLE III

Group		Degrees of freedom	Correlation coefficient	P.
Ash *v.* E	R	11	−0·25	> 0·1 Not significant
	N	18	0·40	> 0·1 Not significant
	S	5	0·10	> 0·1 Not significant
Combined *		34	0·06	> 0·1 Not significant
Combined		36	0·73	< 0·01 Highly significant
Ash *v.* S_B	R	11	−0·05	> 0·1 Not significant
	N	18	0·37	> 0·1 Not significant
	S	5	0·24	> 0·1 Not significant
Combined *		34	0·21	> 0·1 Not significant
Combined		36	0·76	< 0·01 Highly significant
S_B *v.* E	R	11	0·30	> 0·1 Not significant
	N	18	0·76	< 0·01 Highly significant
	S	5	0·98	< 0·01 Highly significant
Combined *		34	0·63	< 0·01 Highly significant
Combined		36	0·90	< 0·01 Highly significant

* Corrected by elimination of effect of diet.

SUMMARY

1. Three groups of one month old rats were fed for a period of four to five weeks on a rachitogenic diet (group R), the same rachitogenic diet with vitamin D (group N), and a complete diet (group S).

2. Young's modulus of elasticity E for bone can be derived from measurements of the deflexion of the centre of a femur loaded at the centre and supported at its ends.

3. The three different diets produced significant differences in breaking stress S_B, Young's modulus E, and percentage ash in the bones. It has not been shown conclusively that higher ash content alone is responsible for the greater S_B and E values of bones produced on the better diets.

4. The value of E in group R was $0{\cdot}6\times10^6$ lb./in.^{2}; in group N $1{\cdot}0\times10^6$ lb./in.^{2}; and in group S (which can be taken as normal) $1{\cdot}6\times10^6$ lb./in.^{2}.

5. There is a high correlation between S_B and E even when the effect of diet is eliminated.

6. Although the bones produced on the good diet (group S) were much stronger than those of groups N or R, the strain at the elastic limit was the same (about 1·5 per cent.). The strain at rupture tended to be higher in groups N and R than in group S.

7. The properties of bone as a structural material are discussed.

We are grateful to Dr I. M. Dawson for allowing us to quote the results of his X-ray examinations. Figure 5 is reproduced by courtesy of the Editors of the Journal of Physiology. We are greatly indebted to Dr W. L. M. Perry for criticising the statistical treatment of our data and for contributing a most useful addendum. The data in this paper may be converted to the metric system from the conversion factor 1000 lb./in.^{2} = 0·703 kg./mm.^{2}.

REFERENCES

BELL, G. H., CHAMBERS, J. W., and DAWSON, I. M. (1947): Journal of Physiology, **106,** 286.
BELL, G. H., and CUTHBERTSON, D. P. (1943): Journal of Endocrinology, **3,** 302.
BELL, G. H., CUTHBERTSON, D. P., and ORR, J. (1941): Journal of Physiology, **100,** 299.
BELL, G. H., and WEIR, J. B. DE V. (1949): Medical Research Council Memorandum No. 22, 85.
DAWSON, I. M. (1946): Nature, **157,** 660.
STEENBOCK, H., and BLACK, A. (1925): Journal of Biological Chemistry, **64,** 263.
Tabulae Biologicae (1925): **1,** 61.
THOMSON, W. (1936): Journal of Hygiene, Cambridge, **36, 24.**

INNERVATION OF THE LIMBS

R. J. LAST, LONDON, ENGLAND

Lecturer in Anatomy, Anatomical Curator, and Bland Sutton Research Scholar, Royal College of Surgeons of England

In this paper the nerve supply of the limbs is considered under three headings: the segmental supply of the skin, the segmental supply of limb muscles, and the double nerve supply of muscles. *Segmental supply of the skin*—No new matter is presented but discussion of the dermatomes is included because restatement of the facts appears to be warranted by the misconceptions shown by many postgraduate students. Moreover, study of the sensory segmental supply provides a convenient introduction to the consideration of motor segmentation to form a basis for comparison with the motor segmental supply. *Segmental supply of limb muscles*—The conceptions here presented are new. The scheme has been used by the author in clinical practice since 1923. That it is more than a mnemonic, and is a sound statement of anatomical fact, will be shown in the discussion. *Muscles with a double nerve supply*—Apparent anomalies in the nerve supply of certain muscles are explained.

SEGMENTAL SUPPLY OF THE SKIN

In all vertebrates the musculature of the body wall has a regular segmental motor innervation that for the most part corresponds with the overlying cutaneous segmental supply. For example, the muscles of an intercostal space are supplied by the same spinal segment that innervates the skin over the space, and the dermatomes and myotomes of the thorax and abdomen are arranged in orderly sequence when traced from the cranial towards the caudal end of the trunk. In the limbs, however, there is no such orderly sequence of segmental innervation, and in no sense could the dermatomes and myotomes be said to correspond.

FIG. 1

The three strips of body wall. The limb buds emerge from the lateral strip (stippled in the diagram) innervated by the lateral branches of the mixed spinal nerves.

Dermatomes—The body wall of all vertebrates is supplied by mixed spinal nerves, arranged in regular segmental sequence. Each mixed spinal nerve after emergence from the intervertebral foramen divides into posterior and anterior primary rami (Fig. 2). The anterior primary ramus ends in its anterior terminal branch near the ventral midline; it gives off a lateral branch which itself divides into anterior and posterior divisions. Thus the body wall is supplied in three longitudinal strips: the extensor muscles of the vertebral column with the overlying skin are innervated from the posterior primary rami; the lateral part of the body wall is supplied by the lateral branches; and the ventral body wall is supplied by the terminal branches of the anterior primary rami. In all vertebrate embryos the limb buds arise from the lateral strip of body wall, supplied by the lateral branches of the anterior primary rami (Fig. 1). Miller and Detwiler (1936) showed that limb muscles probably arise from unsegmented somatopleure mesoderm; but the plurisegmental origin, and number of nerves contributing to the limb plexus of the adult, correspond to the location and cranio-caudal length of the embryonic limb bud. Murray (1928) indicated that the dermis of the limbs arises likewise from the somatopleure.

The lateral branch of each nerve of the limb plexus is projected into the limb to supply it with both sensory and motor fibres. The anterior and posterior divisions of the lateral branch supply the anterior and posterior compartments of the limb respectively.* Thus we find in the roots of all vertebrate limb plexuses a separation into anterior and posterior

* "Anterior" corresponding to "ventral" or "flexor"; and "posterior" to "dorsal" or "extensor."

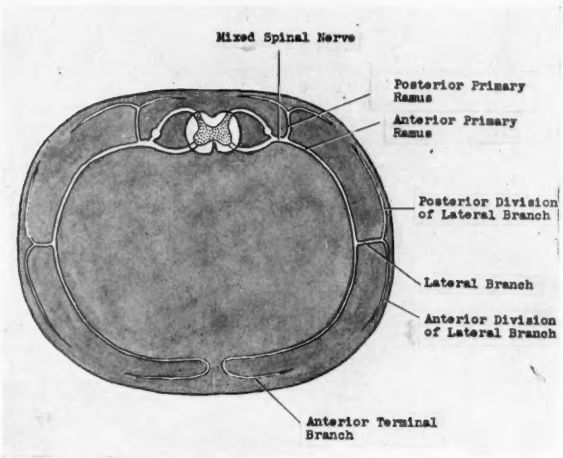


FIG. 2

The distribution of a typical mixed spinal nerve.

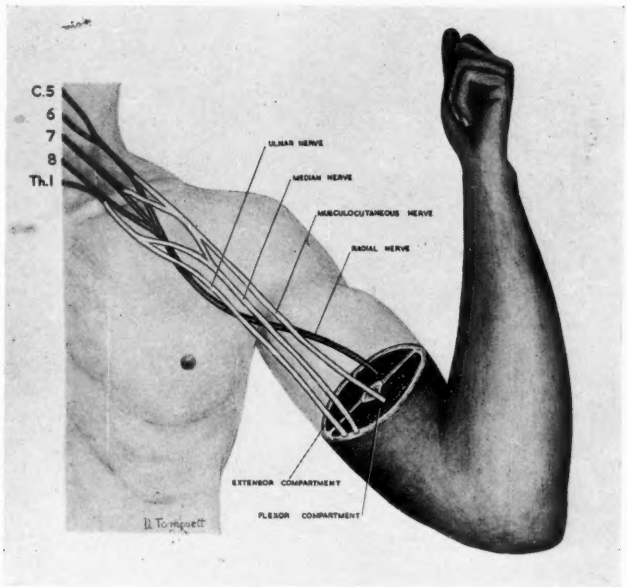


FIG. 3

The destination of the anterior and posterior divisions of the trunks of the brachial plexus to the flexor and extensor compartments of the upper limb.

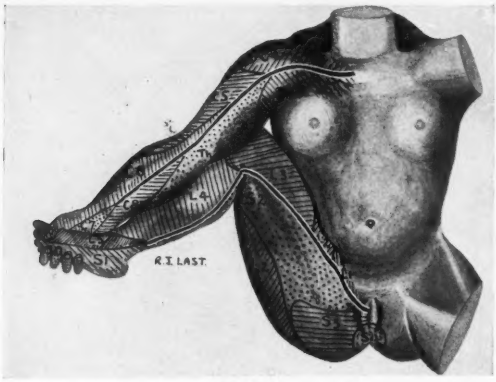


FIG. 4

The dermatomes and anterior axial lines (from a *Figure volante* by Rodin—" Iris Messagère des Dieux ").

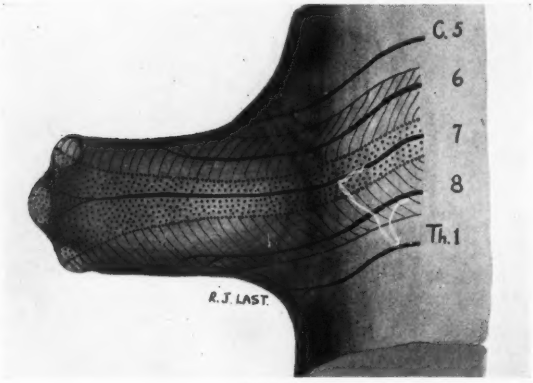


FIG. 5

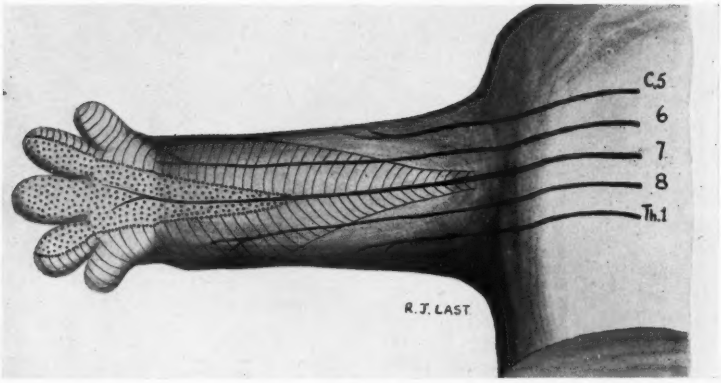


FIG. 6

The innervation of the early forelimb bud. At an early stage C.7 dermatome is still connected with the trunk (Fig. 5). Fig. 6 represents a later stage and shows the early formation of the axial line—C. 7 dermatome is no longer connected with the trunk.

divisions. In the case of the brachial plexus the roots first unite into trunks which themselves divide; in the case of the lumbo-sacral plexus the roots divide first and the divisions unite to form the nerves of the lower extremity. In each case the anterior and posterior divisions provide separate innervation for the flexor and extensor compartments (Fig. 3). Herringham (1886) showed the orderly arrangement by which the skin is supplied on both flexor and extensor surfaces of the limb. He stated his " law " under two headings: 1) Of two spots on the skin, that which is nearer the pre-axial border tends to be supplied by the higher nerve; 2) of two spots in the pre-axial area, the lower tends to be supplied by the lower nerve and of two spots in the post-axial area the lower tends to be supplied by the higher nerve. He continued: " Thus, for example, in the arm C.7 will always tend to supply the parts lying nearest the axis of the limb and farthest from the axis of the body, while the sixth, fifth and fourth in the pre-axial area, and the eighth, ninth and tenth (*i.e.*, C.8, Th.1, Th.2) in the post-axial area, will in that order approach the trunk." Put in another way, Herringham's law stated that progression from pre-axial to post-axial border across the limb encounters the segments in numerical sequence whether the movement is made across the flexor or extensor surfaces of the limb (Fig. 4). For example, according to Herringham, movement from the cephalic to the basilic vein crosses C.5, 6, 7, 8 and Th.1 in that order on both the flexor and extensor surfaces of the limb.

Axial lines—Sherrington (1893) pointed out that Herringham's law breaks down in the proximal area of a limb. On both flexor and extensor surfaces there is a line along which certain dermatomes are missing—a line that represents a gap, across which discontinuous dermatomes meet. This *axial line* is possibly produced in the following way. The central root of the limb plexus is the first to become attached to the growing tip of the limb bud. This is C.7 in the upper limb and L.5 or S.1 in the lower limb. The skin is supplied, at first, by this root alone (Fig. 5). As the limb bud grows, this area of skin is projected distally and is distracted from the trunk, adjacent dermatomes on the cranial and caudal sides moving in to fill the gap (Fig. 6). The line where they meet represents the beginning of an axial line. For example, in the upper limb the central segment of the plexus (C.7) is attached to the growing tip of the limb. Proximally C.6 and C.8 come into contact at the beginning of the axial line; they in their turn become distracted from the trunk, C.5 and Th.1 coming into contact with each other more proximally still. In this way the skin supplied by the five roots is entirely on the limb, which has, indeed, " stolen " some trunk skin cranially (C.4) and caudally (Th.2 and Th.3) as shown in Figure 4.

In the upper limb the anterior axial line extends from just above the wrist to the sternal angle of Louis. At the level of the second costal cartilage the dermatomes C.4 (supraclavicular nerves) and Th.2 are in contact; the first intercostal nerve has no cutaneous branch and the five missing dermatomes have been distracted outwards into the limb. The posterior axial line extends from the vertebra prominens to a point just below the deltoid insertion; the arrangement of dermatomes on the flexor and extensor surfaces of the limb is roughly symmetrical.

In the lower limb there is great asymmetry. This is due partly to extension and rotation of the adult limb from the foetal position of flexion, and partly to the encroachment of trunk skin supplied by Th.12 and L.1 on to the lower extremity. The anterior axial line of the lower limb is mostly on the posterior surface—it is shown in Figure 4 where part of the posterior axial line is also visible on the flexor surface of the thigh. All these facts have been well presented by Harris (1943) and are accepted by most investigators. It seems easiest to summarise the arrangement of the dermatomes by noting their regular segmental sequence if the axial line is traced down to its extremity along its pre-axial side and back along its post-axial side (Fig. 4).

In the upper limb the segments encountered are C.4, 5, 6, 7, 8, Th.1, 2 in that order and in the lower limb L.1, 2, 3, 4, 5, S.1, 2, 3. Adjacent dermatomes overlap considerably, but

there is no overlap across the axial lines. The clinical value of the axial lines is very great in the investigation of root lesions or of segmental lesions of the cord. For accurate localisation of a single root lesion (as, for example, in disc protrusions) it is helpful to be able to test cutaneous anaesthesia or paraesthesia in regions of missing dermatomes—that is, across the axial lines—for here the nerve supply to the skin changes abruptly and there is no confusing overlap of dermatomes such as exists in the rest of the limb and, for that matter, in the trunk itself.

Another well-known application arises in spinal analgesia. A "low spinal" will anaesthetise the skin of the perineum as far forwards as the anterior axial line, the proximal end of which lies at the root of the penis (clitoris). The posterior two-thirds of the scrotum (labium majus) are included, for the dermatome is S.3. To anaesthetise the anterior one-third of the scrotum (labium majus) it is necessary to go seven segments higher to the level of L.1; there are no less than six missing dermatomes across the proximal end of the anterior axial line. They are all out on the limb, covering it with skin (Fig. 4).

For accurate maps of the distribution of dermatomes and the site of the axial lines the reader is referred to "Aids to the Investigation of Peripheral Nerve Injuries," a Medical Research Council pamphlet published by H.M. Stationery Office and reproduced in Cunningham's Text-book of Anatomy (1943). In these maps the overlap of dermatomes is disregarded. For a separate map of each dermatome, ignoring the axial lines, see Foerster (1933) modified in Grant's Atlas (1943).

More recently, Keegan and Garrett (1948) reported their findings from a large series of cases, clinical and experimental. In several respects they disagreed fundamentally with Sherrington's and Foerster's delineation of the limb dermatomes. In particular, according to their evidence the posterior axial lines do not exist: the cranial and caudal roots of each plexus spiral around the pre-axial and post-axial borders from behind to meet at the anterior axial line of the limb. If their findings are confirmed a fundamental alteration of the accepted dermatome maps will be required. On the whole, the dermatomes of Keegan and Garrett are more extensive than those of Sherrington, Head and Foerster. Nevertheless, their findings are open to certain criticisms. 1) The subjective method of mapping a dermatome by hypoalgesia, must be open to wide error. 2) The lack of overlap of adjacent dermatomes is difficult to accept in face of the almost unanimous opinions of countless observers. 3) No mention is made of variability, yet pre-fixation and post-fixation of the plexuses are known to be common. 4) Their claim that an isolated nerve root is affected in their cases of disc protrusions or injected medical students is not convincing; there may well have been some involvement of adjacent nerve roots. Those interested should consult the article of Keegan and Garrett (1948).

SEGMENTAL SUPPLY OF LIMB MUSCLES

The position of the axial lines and dermatomes is well known and easily remembered; but the existence of an orderly sequence in the motor innervation of the limb musculature is not so widely appreciated. In order to learn the segmental origin of the motor nerve to every limb muscle it has hitherto been necessary to memorise long tables which, in themselves, are meaningless. Similarly the muscular distribution of any spinal root of a limb plexus has to be memorised. The lack of correspondence between overlying dermatomes and underlying myotomes has often proved a source of confusion, or at least perplexity, to the student to whom the comparatively orderly arrangement of the dermatomes seems to cover a bewildering chaos of myotomes. But if the fundamental manner in which *joint movements are segmentally innervated* is appreciated the whole of the complex subject of muscle segmentation becomes rational and can be mastered in a matter of minutes.

Rules of segmental innervation—In general it may be said (though this is less true of the upper limb than the lower) that any movement of a joint is innervated by two adjoining segments. The four segments concerned in a movement and its opposite are in numerical

sequence; they control all movements possible in the joint. For example, the spinal centre for the hip joint includes lumbar segments 2, 3, 4, 5, of which 2 and 3 control flexion, medial rotation and adduction, while 4 and 5 control the opposite movements, namely, extension, lateral rotation and abduction. The second rule is that in passing distally one joint into the limb the four segments comprising the joint innervation centre are, *en bloc*, one segment lower in the cord. Thus the centre for the knee is 3, 4, 5, 1 (lumbar and sacral respectively) and it follows that for the ankle, one joint lower in the limb, the centre will be one segment lower, namely, 4, 5, 1, 2.

Lower limb—The spinal centres controlling the hip, knee and ankle consist each of four segments, namely: hip 2, 3–4, 5; knee 3, 4–5, 1; and ankle 4, 5–1, 2. To dissociate the centres into flexor and extensor components, start by contracting the *anterior* muscles first. These are innervated from the upper two segments in the case of each joint (Table I).

TABLE I

SEGMENTAL INNERVATION OF JOINTS OF THE LOWER LIMB

Hip		Knee		Ankle		Foot	
Flex	2						
	3	Extend	3				
Extend	4		4	Dorsiflex	4		
	5	Flex	5		5		
			1	Plantarflex	1	Intrinsic muscles	1
					2		2
							3

These and other joint movements to be described can be performed (if only mentally) while saying the numbers and in this way the whole system can be memorised readily, as in learning the movements of an exercise (Fig. 7). A knowledge of the primary action of any muscle is all that is now required to give its nerve supply. The following are examples:

Psoas and iliacus (flex hip)	2, 3
Vastus intermedius (extends knee)	3, 4
Soleus (flexes ankle)	1, 2

The above are simple antero-posterior movements of the joints of the lower limb. The segmental innervation of inversion and eversion of the foot is shown in Table II.

TABLE II

SEGMENTAL INNERVATION OF TARSAL MOVEMENTS

Invert foot	4
Evert foot	5
	1

This gives an indication of the innervation of the anterior and posterior tibial and the peroneal muscles. It is important to remember this, because tibialis posterior is innervated by L.4, not by S.1 and S.2 like the other foot flexing muscles among which it lies. It should also be noted that all the movements at the hip are innervated by the same four segments that control flexion and extension (Table III).

TABLE III

SEGMENTAL INNERVATION OF LATERAL AND ROTATIONAL MOVEMENT AT THE HIP

Adduction and medial rotation (*cf.* flexion) . .	2
	3
Abduction and lateral rotation (*cf.* extension) . .	4
	5

From this analysis it will be seen that, with the exception of inversion of the foot, each movement of a joint is controlled from two spinal segments, and the opposite movement by the two segments next in numerical sequence. If these movements with their corresponding numbers are memorised it becomes a simple matter not only to state the segmental value of a given muscle but also mentally to work out the essential muscular distribution of any segment.

Upper limb—In the case of the upper limb the rule that each joint movement is controlled through two contiguous spinal segments does not hold quite so universally as in the case of the lower limb. In three cases the essential segment is a single one. But the rule that joints farther down the limb are controlled by spinal centres lower down in the cord still holds. The segments controlling the joints of the upper limb are shown in their simplest form in Table IV.

TABLE IV

SEGMENTAL INNERVATION OF JOINTS OF THE UPPER LIMB

Shoulder	Elbow		Wrist	Hand
Abduct, externally rotate 5	Flex 5			
Adduct, internally rotate 6	6	Pronate 6		
7		Supinate 6	Flex 6	
	Extend 7		7	
8	8		Extend 8	Intrinsic muscles Th.1

It is to be noted that flexion and extension of the shoulder are not included; pectoralis major, latissimus dorsi and deltoid are used in these movements, and they are already covered in the above analysis. The segments involved are 5, 6, 7, 8 (5 for deltoid and 6, 7, 8 for pectoralis major and latissimus dorsi). The following examples indicate the application of the formula:

Pectoralis major (adducts shoulder)	6, 7, 8
Latissimus dorsi (adducts shoulder)	6, 7, 8
Deltoid (abducts shoulder)	5
Subscapularis (internally rotates shoulder)	6, 7
Brachioradialis (flexes elbow)	5, 6

It may be objected that in the first two examples the list of segments quoted is not complete, as the muscles receive fibres from all five roots of the brachial plexus; or it may be pointed out that the deltoid receives C.5 and 6, not merely C.5 as given here. Although muscles may receive fibres from several roots it is usually true that not all these roots are of equal significance. The constitution of the phrenic nerve illustrates the point. There is no doubt that C.3, 4 and 5 form the phrenic nerve, but the essential segment is C.4, for without it the diaphragm will not function. Therefore, for practical purposes the segmental innervation of the diaphragm is C.4, and it is indeed debatable whether it is not poor teaching to include C.3 and 5, in case the student should give them an emphasis equal to C.4. The scheme outlined here is intended to indicate the *essential* segments with sufficient accuracy for clinical purposes. For the clinician and the medical student the scheme provides a simple method of remembering the segmental supply of any limb muscle and the muscular distribution of any spinal segment.

Detailed analysis of segmentation in the gluteal region and forearm—For those who wish to have a more accurate conception of segmental innervation of muscles the gluteal region and forearm may be considered further.

The muscles of the gluteal region—In the simple scheme the innervation of extension, abduction and lateral rotation of the hip is given as L.4, 5. These are the essential segments, but others contribute. Fortunately there is an easy scheme of memorising the more detailed arrangement: it is to remember the superior gluteal nerve as 4, 5, 1 and the inferior gluteal as one segment

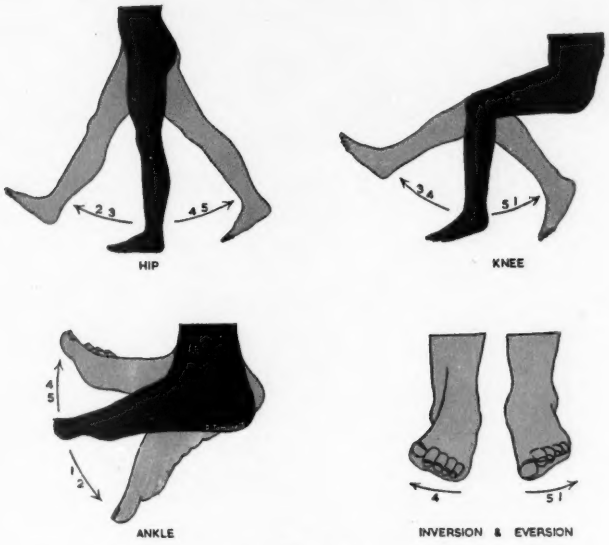


FIG. 7

The segmental innervation of the movements of the lower limb.

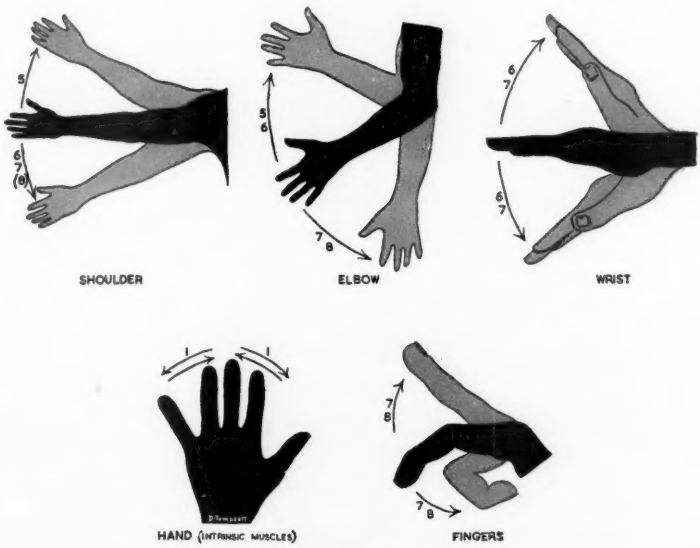


FIG. 8

The segmental innervation of the movements of the upper limb.

lower, namely, 5, 1, 2. The same three segments are contained in the nerve to quadratus femoris and the nerve to obturator internus respectively (note that the higher group of segments supplies the apparently lower muscles). (Table V.)

TABLE V

SEGMENTAL INNERVATION OF THE GLUTEAL MUSCLES

Nerve to quadratus femoris and inferior gemellus	4, 5, 1	Superior gluteal nerve (gluteus medius, gluteus minimus, tensor fasciae femoris)
Nerve to obturator internus and superior gemellus	5, 1, 2	Inferior gluteal nerve (gluteus maximus)

All the muscles of the buttock fall into one or other of these groups except piriformis, which is not a true lower limb muscle but part of the prevertebral rectus that has migrated downwards to gain attachment to the lower limb and still retains its segmental innervation from separate spinal nerves (S.1,2). The lower group, 5, 1, 2, supplies gluteus maximus (inferior gluteal nerve) and obturator internus with its superior gemellus (nerve to obturator internus); all the remaining muscles of the buttock are supplied by 4, 5, 1.

The muscles of the forearm—In the simple scheme the innervation of all the flexor muscles in the forearm is given as C.6, 7; and of all the extensors, C.8. It is probably more important to enlarge this formula than is the case with the muscles of the buttock. The second rule of segmental innervation holds in the case of the forearm, namely, that the more distal joints in the limbs are under control of more caudally placed centres. It is best to consider them from above downwards, remembering the elbow as 5, 6–7, 8 (Fig. 8). The detailed segmental innervation is shown in Table VI.

TABLE VI

SEGMENTAL INNERVATION OF THE MUSCLES OF THE FOREARM

Wrist—Flex	6, 7
Extend	6, 7
Fingers and thumb—Flex	7, 8
Extend	7, 8
(one joint lower, one segment lower)	
Hand intrinsic muscles	Th.1
(one segment lower)	

It is interesting to note the innervation of the wrist flexors. Flexor carpi radialis is innervated by C.6, 7, and this is indicated in the above formula; but it does not appear that flexor carpi ulnaris fits into this scheme, innervated as it is from the ulnar nerve. The medial cord, from which the ulnar nerve is derived, contains C.8 and Th.1 fibres only. But Walsh (1877) has described a contribution from the lateral cord to the ulnar nerve in the axilla in over 90 per cent. of 290 dissections of the brachial plexus, and in half the remainder he found a communication from C.7 to the medial cord and so to the ulnar nerve. Wilfred Harris (1904) in thirty dissections made similar observations; the lateral head of the ulnar nerve was present in twenty-six dissections, and he adds the significant observation that clinical evidence suggests that flexor carpi ulnaris is supplied with C.7 fibres. A frequency of the order found by Walsh and Harris makes the delicate lateral head of the ulnar nerve almost as constant a constituent of the brachial plexus as any other nerve, in spite of its absence from current text-book descriptions. It is the writer's suggestion that the reason for the existence of the lateral head of the ulnar nerve is that flexion of the wrist is under control of the wrist centre at C.6, 7, and that the efferent fibres from this centre must reach flexor carpi ulnaris, and are forced to take this route. The flexor carpi ulnaris is, in fact, innervated by these fibres from the seventh cervical segment.

Discussion—Among the earliest investigations into the segmental supply of limb muscles were those of Ferrier and Yeo (1881), who reported the multi-jointed movements obtained by stimulation of single spinal nerves and determined most of the muscles that contract. They stated "It is evident that the cervical and lumbar enlargements of the spinal cord are centres of highly co-ordinated muscular combinations." It has been said that in no sense can the dermatomes and myotomes of the limbs be said to correspond. There is one minor point of apparent resemblance that can be very misleading: in a few situations a muscle on the pre-axial border has a higher segmental supply than one on the post-axial border (for example, brachio-radialis and the ulnar flexor and extensor of the wrist; the anterior and posterior tibials and the peronei). Such an arrangement is entirely fortuitous and has no place in the underlying plan of limb segmentation. Nevertheless, it was enough to deceive Herringham (1886) who stated: " The superficial thumb muscles are supplied by C.6 or C.7, never from C.8 or Th.1; the little finger muscles are supplied by C.8 as are the deep muscles of the hand. There is no Th.1 in the hand." Sherrington (1892) denied this and showed that, on the contrary, the intrinsic muscles of the hand are supplied by Th.1 only. In the Rhesus monkey he showed, too, that in the muscles of the hand there is no intra-segmental variation in Th.1 segment from the pre-axial to the post-axial side of the hand. Furthermore he pointed out that in the case of the gastrocnemius the lateral head has a higher segmental innervation than the pre-axial medial head—thus putting Herringham's hypothesis out of court. Herringham's conception of higher pre-axial supply was brilliantly true of dermatomes, false of myotomes. His conclusions on root distribution were made by direct dissection in man. Sherrington's conclusions were made by stimulation of single separate spinal nerves in Rhesus and the cat, a procedure which, by the nature of the arrangement of the spinal centres, could never produce the normal regular joint movements (Fig. 9). The author agrees with Wilfred Harris (1904) that " neither of these methods is more than approximately correct when its results are directly applied to the motor distribution of the human plexus." It is an example which illustrates the limitations of the analytical method of experimental investigation. It is, in the writer's opinion, only by the synthetic method of approach that the truth can be arrived at in this case. The writer has attempted to reproduce joint movements in the cat by stimulation of the anterior grey columns, but with very equivocal results, as may be expected. It is not possible to confine the stimulus to one single joint centre. It is by clinical observation in man that the segmental motor distribution can be precisely determined; once done, the extent of the spinal centres can be inferred from this evidence.

Much more accurate information is now available than was possessed by either Herringham or Sherrington, though it should be stated that the final word about the segmental innervation of the limb muscles has by no means been written. Many details of distribution of segmental fibres are still unknown. In particular, the destination of S.3 in the medial popliteal nerve is very uncertain; the author feels that there is significance in Guttmann's (1947) view, based on clinical evidence, that it supplies the intrinsic muscles of the sole of the foot; such a distribution would fit in with the concept that the most peripheral joints in a limb are controlled by the most caudally placed spinal centres. In this connection also Sherrington's work is of interest. In the stately circumlocution of a more spacious age he wrote (1892): " In view of my own experiments the probability amounts in my own thinking almost to certainty that in man the lower limit of the motor outflow to the intrinsic muscles of the foot . . . does often, though not always, extend into the third sacral root."

An appreciation of the principles outlined above shows how orderly is the arrangement by which the muscle segments are distributed in the limbs in regular sequence for consecutive joint movements. The whole system of myotomes and their innervation becomes clear. Area 4 in the prerolandic cortex controls not muscles but joint movements. This control is exercised through spinal centres composed of segments that are arranged in orderly sequence from above downwards, generally in groups of four segments for each joint. It is probable

that the subcortical control of common movements in lower animals is in large measure spinal, and that it exists in such spinal centres as have been described above in man. Sherrington (1892) and Bruce (1901) described the existence of such cell aggregates in the adult spinal cord, and the latter's atlas gives a description of their situation and the number of anterior horn cells in each. Sherrington described three groups of cells in the anterior horn; a medial, an antero-lateral and a postero-lateral. He was of opinion, correctly, that the medial group innervates the muscles of the body wall, the lateral groups the muscles of the limbs. At a meeting of the Physiological Society in 1892 he showed the spinal cord of an adult cat. At C.8 the postero-lateral group of anterior horn cells was much reduced on one side —the side from which the forefoot had been amputated when the animal was less than a month old. His conclusion that the motor cells of the limb muscles lie in the lateral regions of the anterior horns has since received ample confirmation, both clinical and experimental. Romanes (1941 etc.) has shown that spinal centres exist as anatomical entities in foetal man, rabbit and rat, and that the more caudally placed centres are situated more posteriorly in the lateral part of the anterior horn. It seems inconceivable that the centres so precisely described by Romanes can be anything but the spinal centres for joint movements inferred to exist by the author; and a hypothetical reconstruction of the joint centres can be made with some confidence. Such a reconstruction of the lower limb centres in man is shown in Fig. 9. A reconstruction for the upper limb centres in the cervical enlargement would be similar, following the formulae indicated in Tables IV and VI. The boundaries of the joint centres are not precise and sharply demarcated, but shade off as do the boundaries of the cortical centres in Area 4. The main central core of the areas is indicated in the formulae; the centre itself can sometimes be taken as extending a further segment above or below that stated—though not beyond the limit of the plexus itself.

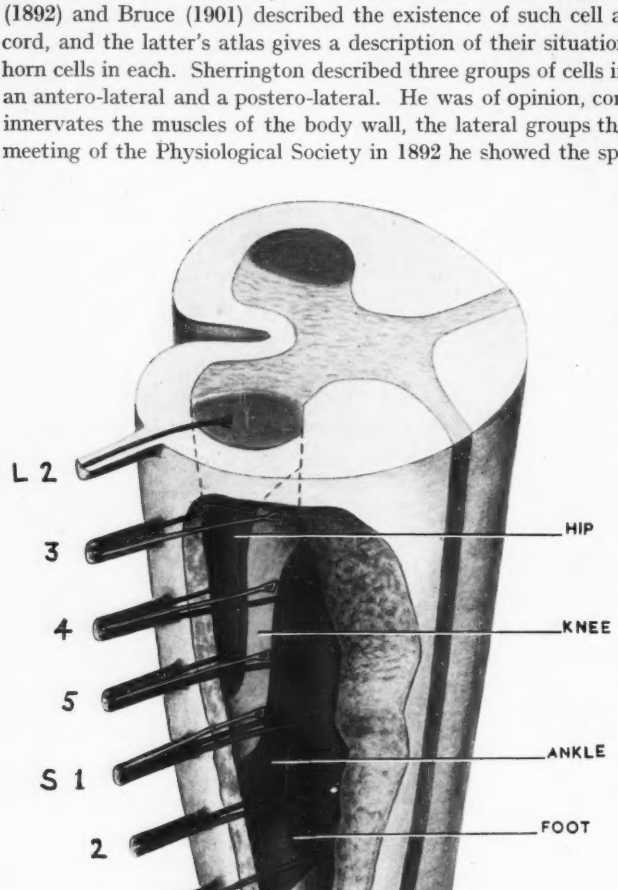


FIG. 9

The spinal centres for the lower limb in man.

The clinical importance of the precise location in the cord of the spinal centres is less than that of the cortical centres, because a lesion confined exclusively to a single joint area

is unlikely to occur in the cord. But the existence of such centres is of great academic interest, and their precise levels can be so easily remembered that the scheme can be made to serve virtually as a mnemonic to place the segmental supply of any muscle. The author has taught the scheme to several hundred postgraduate students, to whom it has given great satisfaction. Many examples of its application have been recalled by these doctors from their own clinical experience. Perhaps the most striking example was the case of a Jamaican member of a class, whose smile of comprehension and satisfaction became wider and wider as the lecture proceeded. At its conclusion the case he presented was his own. Anterior poliomyelitis at the age of eighteen months had left him with gluteal weakness, quadriceps weakness with absent knee jerk, and inability to invert the foot but without foot drop. He had never understood the anatomy of his lesion until that moment—it was a pure L.4 paralysis, as reference to the Tables for the lower limb (Tables I and II) will show.

Pre- and post-fixation—The above figures refer to the segments in normally placed limb plexuses. In cases of pre- and post-fixation the appropriate number of segments must be subtracted or added. The incidence of pre- and post-fixation is high; exact figures are not known. But although a segment to a limb may vary with regard to the vertebral column, the segments always maintain their relative positions to each other, a fact which was known to Herringham in 1886.

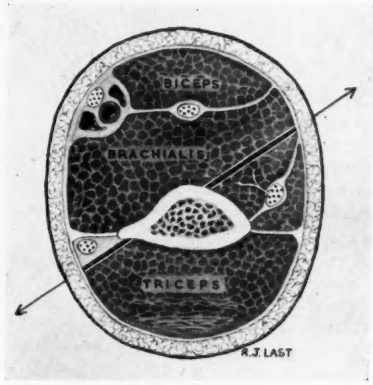


FIG. 10

Cross-section of the arm above the elbow. The arrow indicates the site of the foetal intermuscular septum between flexor and extensor compartments.

MUSCLES WITH A DOUBLE NERVE SUPPLY

Each component root of a limb plexus divides into an anterior and posterior division for the innervation of the flexor and extensor compartments respectively. In the brachial plexus the division takes place after the formation of the trunks, while in the simpler lumbo-sacral plexus it takes place before the formation of the branches. The more precisely acting flexors demand a richer innervation than the coarser extensor muscles; consequently we find that the most caudal root of a plexus (Th.1; S.3) is distributed, as far as its motor fibres are concerned, entirely to the flexor compartment of the limb.

With these facts in mind it is easy to understand that such a muscle as flexor digitorum profundus with its accompanying lumbricals can have a double nerve supply, from the ulnar and median nerves, for each of these is a nerve of the flexor compartment of the upper limb, derived originally from the anterior divisions of the trunks. It is more difficult to understand the branch to brachialis from the radial (musculospiral) nerve, for the radial nerve is the nerve of tne extensor compartment while brachialis is a flexor muscle. The explanation is that the portion of brachialis innervated by the radial nerve developed in the extensor compartment of the foetal limb. Subsequent alteration in position of the septum between the flexor and extensor compartments has included this part of brachialis in the flexor compartment, where it fuses with that part innervated by the musculocutaneous nerve (Fig. 10). Also included in the flexor compartment are the brachio-radialis muscle and that part of the radial nerve which runs above and anterior to the elbow joint. A similar extent of the ulnar nerve, a nerve of the flexor compartment, is contained in the extensor compartment. The lateral and medial intermuscular septa of the adult arm do not represent the foetal division between flexor and extensor compartments.

In the case of the hamstrings, all are supplied by the medial popliteal moiety of the sciatic nerve (the nerve of the flexor compartment) except the short head of the biceps. This, although a flexor muscle, is supplied by the lateral popliteal component of the sciatic nerve, which is the nerve of the extensor compartment of the leg. The reason is that the short head of the biceps was developed in the extensor compartment and only later migrated into the flexor compartment. Pectineus affords another example. An adductor of the thigh, it is always developed in the extensor compartment and is supplied usually by the femoral nerve, the nerve of the extensor compartment; but in one out of three individuals it derives its supply also from an accessory obturator nerve. The obturator nerve itself is a nerve of the flexor compartment. But the accessory obturator nerve is misnamed; it arises from *posterior* divisions of the lumbar nerves (2, 3) and notwithstanding the fact that it emerges on the medial side of psoas should rightly be called the accessory femoral. Like the femoral it emerges on the cephalic side of the pubic bone; the true obturator nerve emerges on the caudal side of that bone. In some cases pectineus receives fibres from the true obturator nerve; in such cases that part of the muscle innervated from the obturator will have developed in the flexor (obturator) compartment. The muscle lies at the pre-axial border of the limb and, like brachialis, is formed by fusion of flexor and extensor flesh.

In short, the nerve supply of all muscles is a certain guide to their embryological development.

REFERENCES

BRASH, J. C., and JAMIESON, E. B. (1943): Cunningham's Text-book of Anatomy. Eighth edition. London: Humphrey Milford, Oxford University Press. p. 1037, Fig. 905; p. 1061, Fig. 918.

BRUCE, A. (1901): A Topographical Atlas of the Spinal Cord. London: Williams & Norgate.

FERRIER, D., and YEO, G. F. (1881): "The Functional Relations of the Motor Roots of the Brachial and Lumbo-Sacral Plexuses." Proceedings of the Royal Society of London, **32,** 12.

FOERSTER, O. (1933): "The Dermatomes in Man." Brain, **56,** 1.

GRANT, J. C. B. (1943): An Atlas of Anatomy. Baltimore: William Wood & Company. Figs. 14 and 156.

GUTTMANN, L. (1947): Stoke Mandeville Hospital, Bucks. Personal Communication to the Author.

HARRIS, H. A. (1943): "The Anatomical Basis of Physical Medicine with Special Reference to the Peripheral Nervous System." Proceedings of the Royal Society of Medicine, **36,** 299.

HARRIS, W. (1904): "The True Form of the Brachial Plexus and its Motor Distribution." Journal of Anatomy and Physiology, **38,** 399.

HERRINGHAM, W. P. (1886): "The Minute Anatomy of the Brachial Plexus." Proceedings of the Royal Society of London, **41,** 423.

KEEGAN, J., and GARRETT, F. (1948): "The Segmental Distribution of the Cutaneous Nerves in the Limbs of Man." Anatomical Record, **102,** 409.

MEDICAL RESEARCH COUNCIL. War Memorandum No. 7 (1943): "Aids to the Investigation of Peripheral Nerve Injuries." H.M. Stationery Office, London.

MILLER, RUTH A., and DETWILER, S. R. (1936): "Comparative Studies upon the Origin and Development of the Brachial Plexus." Anatomical Record, **65,** 273.

MURRAY, P. D. F. (1928): "The Origin of the Dermis." Nature, **122,** 609.

ROMANES, G. J. (1941): "Cell Columns in the Spinal Cord of a Human Foetus of Fourteen Weeks." Journal of Anatomy, **75,** 145.

ROMANES, G. J. (1941): "The Development and Significance of the Cell Columns in the Ventral Horn of the Cervical and Upper Thoracic Spinal Cord of the Rabbit." Journal of Anatomy, **76,** 112.

ROMANES, G. J. (1942): "The Spinal Cord in a Case of Congenital Absence of the Right Limb below the Knee." Journal of Anatomy, **77,** 1.

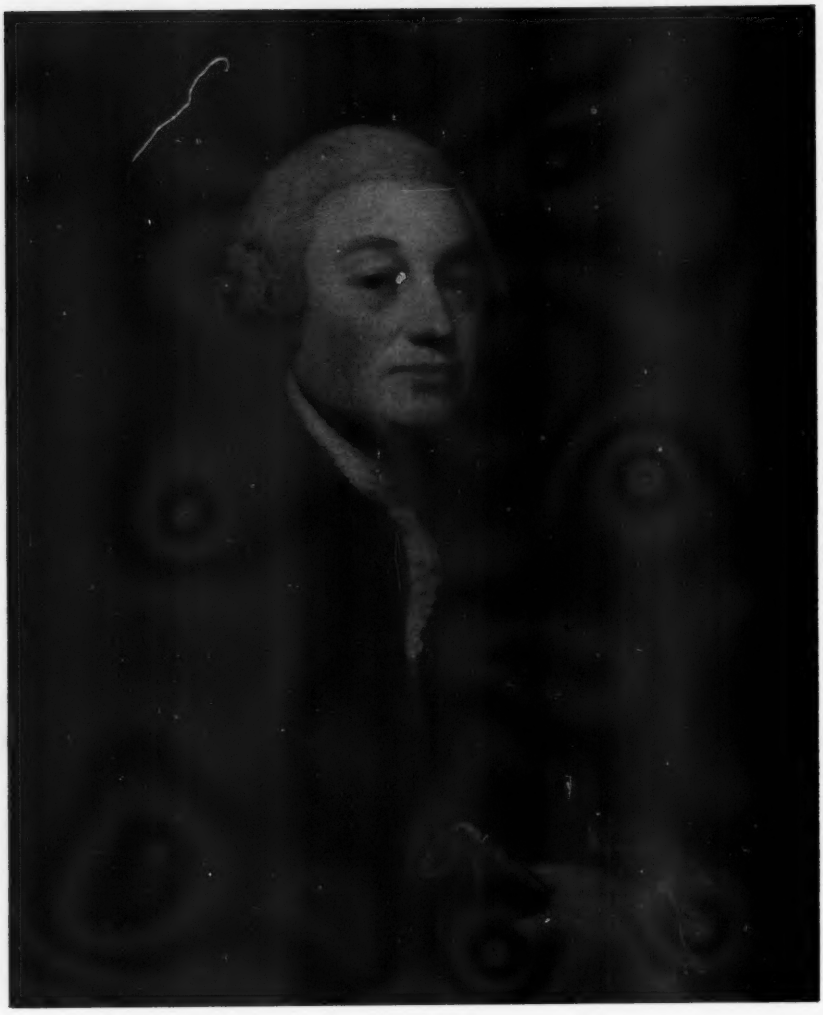
ROMANES, G. J. (1946): "Motor Localisation and the Effects of Nerve Injury on the Ventral Horn Cells of the Spinal Cord." Journal of Anatomy, **80,** 117.

SHERRINGTON, C. S. (1892): "Notes on the Arrangement of some Motor Fibres in the Lumbo-Sacral Plexus." Journal of Physiology, **13,** 621.

SHERRINGTON, C. S. (1893): "Experiments in Examination of the Peripheral Distribution of the Fibres of the Posterior Roots of some Spinal Nerves." Philosophical Transactions of the Royal Society of London (B) **184,** 641.

SHERRINGTON, C. S. (1898): "Experiments in Examination of the Peripheral Distribution of the Fibres of the Posterior Roots of Some Spinal Nerves, Part 2." Philosophical Transactions of the Royal Society of London, (B) **190,** 45.

WALSH, J. (1877): "The Anatomy of the Brachial Plexus." American Journal of the Medical Sciences, **74,** 387.



Percivall Pott
1714-1788

Percivall Pott

Percivall Pott is perhaps the best-known English surgeon of the pre-antiseptic era. His fame in the eighteenth century has persisted and been maintained by clear descriptions of the injury and diseases of bone that are associated with his name. In him is to be seen the beginning of an attitude untrammelled by irrational obedience to the dictates and practices of the early fathers of medicine. He had great influence on the development of English surgery.

He was born on January 6, 1714, in Threadneedle Street, London. The house was subsequently pulled down and on its site an extension of the Bank of England was built. His father, a descendant of an old Cheshire family, died when he was only three years old, leaving a wife and child in somewhat straitened circumstances. The mother, anxious about the boy's education, received help from her relative, Dr Wilcox, Bishop of Rochester, and Percivall was thus sent to a school at Darenth in Kent. Here he made good progress in the classics and it was thought that he might become a candidate for holy orders; but he was attracted to medicine.

To secure entrance to the medical profession, apprenticeship to a regular practitioner was then necessary and most pupils became attached to an apothecary in private practice. Few probationerships were available at hospitals but young Pott was fortunate, for in his sixteenth year he obtained a seven years' apprenticeship to Edward Nourse, assistant surgeon to St. Bartholomew's Hospital, paying two hundred guineas for his indentures. Nourse lectured in anatomy and surgery at Barber-Surgeons' Hall and at London House in Aldersgate Street. For these lectures Pott dissected demonstration specimens and laid the foundation of the anatomical knowledge which later gave him so great an advantage over his contemporaries.

After apprenticeship to Edward Nourse, on "September 7, 1736, Percivall Pott was admitted to the Freedom of the Company (of the Barber-Surgeons) by service, upon the testimony of his master and was sworn." Later the same day "the said Mr Percivall Pott was examined touching his skill in surgery in order to have the Great Diploma. His answers were approved, and he was ordered a Diploma under the seal of the Company and the hands of the Governors testifying his skill and impowering him to practise." The Great Diploma was a rare award and was granted only after very thorough examination; in some ways it corresponded to the present F.R.C.S.

Pott took a house in Fenchurch Street into which he moved with his mother and her daughter by her first marriage. A few years later he moved to Bow Lane and whilst practising there took the livery of the Barber-Surgeons' Company and paid the usual fine of £10. In 1745 he was elected assistant surgeon to St. Bartholomew's Hospital, becoming full surgeon four years later.

In the year that Pott was appointed to the staff of St. Bartholomew's, the Barber-Surgeons' Company was dissolved by Act of Parliament after a partnership of two hundred years. A few weeks after separating, the surgeons met together at Stationers' Hall as "The Master, Governors and Commonality of the Art and Science of Surgery" which body afterwards became known as the Corporation of Surgeons. In 1751 they settled in their own quarters in the Old Bailey. Pott took a very active part in the affairs of the new Corporation and on July 5, 1753, its Court of Assistants elected him and William Hunter as the first Masters (or Lecturers) of Anatomy. Later Pott was appointed to other offices and in 1765 was elected Master (or Governor) of the Corporation.

When Pott began his work as hospital surgeon there was little organised teaching of medical students in London. Samuel Sharp of Guy's gave a course of evening lectures on anatomy, surgical operations and bandaging to a Society of Naval Surgeons which met at

Covent Garden; and Edward Nourse gave occasional lectures on surgical principles at St. Bartholomew's. Percivall Pott was the first to introduce regular teaching of clinical surgery at the bed-side. He spoke of cures, mistakes and experience of other patients with similar disorders and such instruction drew many students around him, some of whom included John Hunter, Abernethy, Blicke and Earle. He also gave lectures in his own house in Watling Street, to which he had removed from Bow Lane, and the attractive manner of his delivery was testified by Sir William Blizzard when he said: " It was difficult to give an idea of the elegance of his language, the animation of his manner or the perceptive force or effect of his truths and his doctrines."

At the time that Pott was elected to the staff of St Bartholomew's Hospital he wrote a paper—" An Account of Tumours which rendered the Bones Soft "—which was published in the Philosophical Transactions. After that contribution he was silent for twelve years, but at the age of forty-three an event occurred which induced him to become a constant writer in surgery whereby he gained world-wide fame. It was in 1756 while riding in what is now known as the Old Kent Road that an accident befell him. Sir James Earle, his son-in-law and biographer, relates that

" He was thrown from his horse, and suffered a compound fracture of the leg, the bone being forced through the integuments. Conscious of the dangers attendant on fractures of this nature, and thoroughly aware how much they may be increased by rough treatment, or improper position, he would not suffer himself to be moved until he had made the necessary dispositions. He sent to Westminster, then the nearest place, for two Chairmen to bring their poles; and patiently lay on the cold pavement, it being the middle of January, till they arrived. In this situation he purchased a door, to which he made them nail their poles. When all was ready, he caused himself to be laid on it, and was carried through Southwark, over London-bridge, to Watling-Street, near St Paul's, where he had lived for some time—a tremendous distance in such a state! I cannot forbear remarking, that on such occasions a coach is too frequently employed, the jolting motion of which, with the unavoidable awkwardness of position, and the difficulty of getting in and out, cause a great and often a fatal aggravation of the mischief. At a consultation of surgeons, the case was thought so desperate as to require immediate amputation. Mr Pott, convinced that no one could be a proper judge in his own case, submitted to their opinion; and the instruments were actually got ready, when Mr Nourse, who had been prevented from coming sooner, fortunately entered the room. After examining the limb, he conceived there was a possibility of preserving it: an attempt to save it was acquiesced in, and succeeded. This case, which Mr Pott sometimes referred to, was a strong instance of the great advantage of preventing the insinuation of air into the wound of a compound fracture; and it probably would not have ended so happily, if the bone had not made its exit, or external opening, at a distance from the fracture; so that, when it was returned into the proper place, a sort of valve was formed, which excluded air. Thus no bad symptom ensued, but the wound healed, in some measure, by the first intention."

Sir D'Arcy Power thought that " the accident which Pott sustained was an open fracture of the tibia—spiral or very oblique—and that the nib-shaped end of the upper fragment penetrated the skin." Bearing in mind the gloomy fate of a compound fracture up to the mid-Victorian era, Pott himself contributed greatly to the preservation of his limb and the good healing of his fracture by his foresight in safeguarding the leg from the moment of accident until he reached his home.

Up to the time of his accident Pott had recorded his experiences and investigations in the manuscripts of his lectures but had published none of them. He took advantage of the leisure imposed by convalescence in preparing for publication and, once started as a writer, continued writing for over twenty years. His first work—" A Treatise on Ruptures "—appeared in 1756, followed by several others on diseases of the testicle, head injuries, curvature of the spine with lower limb palsy, fractures and dislocations.

Pott's Fracture—One of the important contributions to surgery by Pott was his monograph entitled " Some few General Remarks on Fractures and Dislocations " published in 1769. He opposed the existing treatment by continuous instrumental traction which was irksome and fatiguing. He asserted that a fracture could be best reduced and correction maintained

by keeping the limb in such a posture that the muscles were continually relaxed. This teaching had a far-reaching effect, for Pott's method of treating fractures was generally adopted in England and it prevailed for several generations. In this monograph he also described the fracture-dislocation of the ankle that now bears his name, with an illustration of the resulting valgoid displacement of the foot and a drawing of the skeletal injuries responsible for it. His description is quite impersonal and he makes no mention of the fracture that he himself sustained. In consequence there has been some misapprehension as to the nature of Pott's accident. His classical description of the ankle fracture-dislocation, and his reticence about his own fracture of the tibia at a higher level, have misled many to believe that in describing the ankle injury he was speaking of something within his own intimate experience. This misconception has helped to fasten his name to the fracture-dislocation which he portrays in these words:

" When by leaping or jumping the fibula breaks in the weak part already mentioned, that is within two or three inches of its lower extremity. When this happens, the inferior fractured end of the fibula falls inward toward the tibia, that extremity of the bone which forms the outer ancle is turned somewhat outward and upward and the tibia having lost its proper support and not being of itself capable of steadily preserving its true perpendicular bearing, is forced off from the astragalus inwards by which means the weak bursal, or common ligament of the joint is violently stretched, if not torn, and the strong ones which fasten the tibia to the astragalus and os calcis are always lacerated, thus producing at the same time a perfect fracture and a partial dislocation, to which is sometimes added a wound in the integument, made by the bone at the inner ancle. When this accident is accompanied as it sometimes is by a wound of the integuments of the inner ancle and that made by the protrusion of the bone, it not infrequently ends in a fatal gangrene unless prevented by timely amputation though I have several times seen it do very well without. But in its most simple state, unaccompanied with any wound, it is extremely troublesome to put to rights, still more so to keep it in order and unless managed with address and skill, is very frequently productive both of lameness and deformity ever afterward."

Pott's Disease—The best known of Pott's contributions to surgery was his treatise entitled " Remarks on that kind of Palsy of the lower limbs which is frequently found to accompany A Curvature of the Spine and is supposed to be caused by it." It was published in 1779 and was translated into French and Dutch; the disease which it described became known on the continent as " La maladie du Pott." This monograph reveals his ability as a clinical observer and the lucidity of his diction. He painted these patients with their symptoms and signs with so sure a touch that we can add nothing to the picture. He differentiated between flaccid and spastic paralysis and noted that spasticity was the invariable rule of spinal cord pressure in spinal caries. He said:

" The disease of which I mean to speak, is generally called a palsy, as it consists in a total or partial abolition of the power of using, and sometimes of even moving the lower limbs, in consequence, as is generally supposed, of a curvature of some part of the spine. To this distemper both sexes, and all ages, are equally liable. . . . Until the curvature of the spine has been discovered, it generally passes for a nervous complaint. . . . I have in compliance with custom called the disease a palsy . . . yet there are some essential circumstances in which this affection differs from a common nervous palsy: The legs and thighs are rendered unfit for all the purposes of locomotion and do also lose much of their sensibility, but they have neither the flabby feel, which a truly paralytick limb has, nor have they that seeming looseness at the joints, nor that total incapacity of resistance, which allows the latter to be twisted in almost all directions; on the contrary the joints have frequently a considerable degree of stiffness, particularly the ancles, by which stiffness the feet of children are generally pointed downward, and they are prevented from setting them flat upon the ground."

A second essay was published in 1782 in which Pott dealt mainly with the morbid anatomy of disease of the spine, accompanied by engravings illustrating the changes that occurred in the vertebrae. He concluded that the disorder had its origin elsewhere in the body: the disease was scrophula, and was capable of revealing itself in a variety of organs. To give it a modern terminology, tuberculosis is an infective disease with local manifestations. In concluding his essay with a summary of the morbid anatomy he said:

"This morbid affection shews itself in a variety of forms, but although its appearances be various, yet they are always such as determine the true nature of the distemper. Sometimes it appears in a thickened state of the ligaments, connecting the vertebrae together, without any apparent affection of the bones. Sometimes in the form of a distempered state of the intervertebral substances, called cartilages. Sometimes in that of diseased glands, either in a merely indurated and enlarged state or, what is more frequent, in that of a partial suppuration. Sometimes it is found in the form of bags or cysts, containing a quantity of stuff of a unequal consistence, partly purulent, partly sanious, and partly a curd-like kind of substance; and not unfrequently entirely of the last. Sometimes under these bags, or cysts, even while they remain whole, the subjacent bones are found to be distempered, that is deprived of periosteum, and tending to become carious. Sometimes these collections erode the containing membranes, and make their way downward by the side of the psoas muscle, towards the groin, or by the side of the pelvis behind the great trochanter, or in some cases to the outside of the upper part of the thigh. . . . That the disease which produces these effects on the spine, and the parts in its vicinity, is what is in general called the scrophula; that is, that same kind of indisposition as occasions the thick upper lip, the tedious obstinate ophthalmy, the indurated glands under the chin, and in the neck, the obstructed mesentery, the hard dry cough, the glairy swellings of the wrist and ancles, the thickened ligaments of the joints, the enlargement, and caries of the bones."

The treatment of spinal disease had been directed towards the straightening of the kyphosis and was attempted by means of "steel stays, the swing, the screw chair and other pieces of machinery." Pott had observed that no permanent good purpose had been served by these procedures and he deliberately made no attempt to correct the deformity. This was a new departure in treatment and was the first sign of understanding of the natural process of cure by osseous fusion through vertebral collapse. But he was persuaded, partly by the inspiration of Hippocratic teaching, to form an artificial sinus by applying caustic to the skin on each side of the gibbus in the belief that a prolonged flow of exudate had curative value. He seemed confirmed in his view by the frequent relief of paralysis in patients submitted to this operation. It was not performed with the object of draining an abscess, and indeed there seldom is any superficial abscess in Pott's paraplegia. But he did cure the patients in another way. The artificial sinus imposed recumbency, and in consequence of prolonged rest the paralysis disappeared. Pott, like many of his successors, failed to realise the decisive importance of rest. It was not until nearly a century later that the value of rest in joint tuberculosis was formulated by Hilton and Hugh Owen Thomas.

Pott's Puffy Tumour—Pott took considerable interest in head injuries. In 1760 he published a monograph entitled "Observations on the Nature and Consequences of Wounds and Contusions of the Head, Fractures of the Skull, Concussions of the Brain, etc." This was followed in 1768 by another monograph, and two further editions of the work appeared later. These productions were prepared carefully and bore evidence of extensive reading of Latin and French writings on the subject. He did much to simplify trephining of the skull and advanced the knowledge of the morbid anatomy of cerebral injury. His publications included abundant case histories which are interesting apart from their main purpose; his delightful narrative touches upon the occupations, social habits and customs of ordinary people in the eighteenth century. The particular scalp swelling or puffy tumour that he described is referred to in this paragraph:

"If the symptoms of pressure, such as stupidity, loss of sense, voluntary motion, etc., appear some few days after the head has suffered injury from external mischief, they do most probably imply an effusion of a fluid somewhere; this effusion may be in the substance of the brain, in its ventricles, between its membranes, or on the surface of the dura mater; and which of these is the real situation of such extravasation is a matter of great uncertainty; none of them being attended with any peculiar mark, or sign that can be depended upon, as pointing it out precisely; but the inflammation of the dura mater, and the formation of matter between it and the skull, in consequence of contusion, is generally indicated and preceded by one which I have hardly ever known to fail; I mean a puffy, circumscribed, indolent tumour of the scalp, and a spontaneous separation of the pericranium, from the skull under such tumour. These appearances therefore following a smart blow on the head, and attended with languor, pain, restlessness, watching, quick pulse, headache, and slight irregular shiverings, do almost infallibly indicate an inflamed dura mater, and pus, either forming or formed between it and the cranium."

Pott's contributions to the knowledge of head injuries did much to establish him as one of the leading surgeons of his day. But apart from these familiar eponymous disorders, a mass of scientific knowledge deriving from Pott has long since been incorporated in surgical literature. One instance is chimney-sweep's cancer which he was the first to describe; he was the first to point out the carcinogenic properties of soot on man. The experimental verification of Pott's observations on the production of cancer in mice by soot irritation was accomplished by Passey in 1920. Moved by the misery of the chimney-boys he drew the attention of profession and public to the evil nature of their occupation:

" The fate of these people seems singularly hard; in their early infancy, they are most frequently treated with great brutality and almost starved with cold and hunger; they are thrust up narrow and sometimes hot chimneys where they are bruised, burned and almost suffocated; and even when they get to puberty become peculiarly liable to a most noisome, painful and fatal disease."

The employment of chimney-boys was eventually made illegal by Act of Parliament. It is almost incredible that even to-day there should exist a link with this degrading custom but a centenarian still lives who at the age of twelve worked fifteen hours a day, climbed the insides of chimneys, and swept down soot with a hand brush.

The humane disposition of Percivall Pott was displayed in other ways. Before he joined the staff of St. Bartholomew's, extensive use was made of escharotics and the actual cautery, but Pott condemned the practice and ultimately succeeded in abolishing it. Furthermore, he contrived to render surgical treatment as mild as possible, consistent with efficiency; and this principle was reflected in his use at operations of a reduced number of instruments of simple design. These reforms were greeted with some contempt by his colleagues who were accustomed to elaboration of technique but Abernethy, a warm admirer, testified to Pott's consideration for the ease and comfort of his patients.

He also had a kindly heart towards his dressers, some of whom he took into his own home. He took a leading part in improving the instruction of students. His lectures were open to all on payment of a small fee and they were well attended. He facilitated the diffusion of surgical instruction by selling his own publications at low cost instead of in the conventional form of heavy and expensive volumes. His monograph on palsy of the lower limbs in spinal curvature consisted of eighty-three pages and cost one shilling and sixpence, and this venture paved the way for cheap medical text-books.

Judging by portraits of Pott he had a pleasing appearance, and dressed according to the fashion of the period, visiting the hospital in his powdered wig, red coat and buckled sword. In the words of Earle he was " elegant, lower than middle size." He was an excellent conversationalist with ready wit and a fund of anecdotes. He was a devoted son, and made a home for his mother until her death in 1746, after which he married the daughter of Robert Cruttenden by whom he had five sons and four daughters. In 1769 he bought a house near Lincoln's Inn Fields and resided in it for seven years when he moved to Prince's Street, Hanover Square. At this time Sir Caesar Hawkins, who was reputed to have the best surgical practice in London, retired and Pott succeeded him in professional favour.

For the next ten years Pott was much in demand as a consultant and apart from his hospital work he kept up a large correspondence with surgeons and practitioners who sought his opinion and advice from all over the world. He was the recipient of many distinctions: in 1764 he was elected a Fellow of the Royal Society; the next year he was appointed Master of the Corporation of Surgeons; in 1786 he was elected the first Honorary Fellow of the Royal College of Surgeons of Edinburgh and the year after that an Honorary Member of the Royal College of Surgeons in Ireland. These last two honours were conferred upon him at about the time of his retirement from St Bartholomew's Hospital on July 12, 1787, after having, as he said, " served it man and boy for half a century." At the annual meeting of the Hospital subscribers he was elected a Governor and at the dinner that followed there

was a moving scene. The Right Honourable Thomas Harley proposed the toast of Percivall Pott who was usually composed and eloquent but on this occasion was overcome with such emotion that after rising to reply was unable to speak and resumed his seat in silence.

He continued to practise but his retirement lasted only about eighteen months. On December 27, 1788, he died of pneumonia due to a chill which he caught whilst visiting a patient in severe weather twenty miles from London. His last conscious words were: "My lamp is almost extinguished; I hope it has burnt for the benefit of others." He was buried at Aldermary Church in Bow Lane, close to the remains of his mother.

Percivall Pott was a great leader in surgery who shone as a clinical surgeon. He flourished before the emergence of surgical pathology under John Hunter, and the deductions from his clinical observation suffered from this lack of scientific interpretation. He was, however, particularly free from the shackles of tradition and was bold enough to cut a path of his own. In a sense he was more acquainted with the practice of surgery than Hunter but he lacked, as they all lacked before the coming of Pasteur and Lister, the one key that saved surgery from being a tragic adventure.

Percivall Pott is an outstanding figure in the evolution of surgery in Britain. He took part in the formation of the Corporation of Surgeons and became its Master, started organised teaching of medical students, and by his humane attitude, good sense and personal integrity helped greatly to raise the status of surgery in this country. His writings were clear and composed with scholarly grace, and his observations recorded faithfully without being tedious. Their translation into European languages did much to promote the prestige of British surgery abroad.

ARTHUR ROCYN JONES.

REFERENCES

HORDER, T. J. (1894): Life and Works of Percivall Pott. St. Bartholomew's Hospital Reports, **30,** 163.
LLOYD, G. MARNER (1933): Life and Works of Percivall Pott. St. Bartholomew's Hospital Reports, **66,** 291.
POTT, PERCIVALL (1790): The Chirurgical Works of Percivall Pott, F.R.S., to which are added a short Account of the Life of the Author, by James Earle, Esq. Three volumes. London: printed for J. Johnson.
POWER, Sir D'ARCY (1923): Percivall Pott: His own Fracture. British Journal of Surgery, **10,** 313.
POWER, Sir D'ARCY (1929): The Works of Percivall Pott. British Journal of Surgery, **17,** 1.

[This signature is reproduced from a letter, preserved in the library of the Royal College of Surgeons of England, in which Pott wrote concerning the apprenticeship and indentures of his son. We are grateful for permission to reproduce it, and also for permission to reproduce one of the two portraits of Percivall Pott which hang in the Council Room of the Royal College of Surgeons.—EDITOR.]

OTTO KAHLER (1849-1893)

A Centennial Note

How often is there not an element of incongruity, and even of ingratitude, in the way in which the halo of eponymous commemoration encompasses a medical name? That rare and fatal disease, multiple myelomatosis, is known universally in the literature as *Kahler's disease*, in honour of the Prague physician who described it in 1889. For the sake of historical accuracy it may be recalled that this condition was first brought to the notice of the profession in 1846 by John Dalrymple, F.R.C.S., surgeon to the Royal Ophthalmic Hospital, Moorfields, London, in a communication: " On the Microscopical Character of Mollities Ossium " (Dublin Quarterly Journal of Medical Science, 1846, N.S. **2,** 85–95). It is recorded that " no suspicion of the affection of the bones existed during the life of the patient and the mollities was only revealed by the post-mortem examination." The patient was attended by William MacIntyre, M.D., physician to the Western General Dispensary. The urine was examined by Henry Bence Jones, F.R.S., physician to St George's Hospital. Four years later MacIntyre reported this " Case of Mollities and Fragilitas Ossium, accompanied with urine strongly charged with animal matter," in Medico-Chirurgical Transactions (1850, **33,** 211–32). Bence Jones described his test for what is now called " Bence Jones protein " in a paper " On a new substance occurring in the Urine of a patient with Mollities Ossium," which appeared in the Philosophical Transactions of the Royal Society of London, 1848, **138,** 55–62.

In the centennial year of his birth it is appropriate to note the part played by Otto Kahler in the history of multiple myelomatosis.[1] Born in Prague on January 8, 1849, the son of a doctor, he studied medicine in his native city and did post-graduate work in Paris under Charcot and Duchenne, from whom he imbibed a lifelong interest in neuropathology. In 1878 he became *Dozent* at the German University in Prague, and in 1886 Professor of Special Pathology and Therapy. Three years later he succeeded Heinrich Bamberger in the corresponding chair at Vienna. The story of his life is a sad one. Shortly before going to Vienna he noticed a small lump in his tongue which was removed by Carl Gussenbauer but soon recurred. A second unsuccessful operation was performed by Billroth in 1890. There were widespread metastases, leading to his death on January 24, 1893, when at the height of his fame, a few weeks after his forty-fourth birthday.

Much valuable work was crowded into the few years of Kahler's professional activity. He gave the first complete description of syringomyelia in 1888 (Prager medizinische Wochenschrift, 1888, **13,** 45, 63), and in the next year published his paper " Zur Symptomatologie des multiplen Myeloms " in Wiener Medizinische Presse, 1889, **30,** 209–13, 253–5—originally delivered as a lecture before the Verein deutscher Aerzte in Prague on January 11, 1889.[2] His patient was a doctor aged forty-six years. It is interesting to note that the author described the presence of albuminuria and the peculiar nature of the protein. It is unlikely that at that time physicians in Europe were familiar with MacIntyre's and Dalrymple's accounts. Kahler was an excellent clinician, a popular consultant, a lucid and stimulating teacher, and an aristocratic personality with a delightful sense of humour. For several years he edited the Zeitschrift für klinische Medicin and the Prager medizinische Wochenschrift.

W. R. BETT.

[1] The term " *Multiples Myelom* " was first used in 1873 by J. von Rustizky (Deutsche Zeitschrift für Chirurgie, 1873, **3,** 162–72).

[2] This paper also appeared in Prager medizinische Wochenschrift, 1889, **14,** 33, 45, but this I have not seen.

DISMEMBRING

WILLIAM BROCKBANK and D. LL. GRIFFITHS, MANCHESTER, ENGLAND

From the Medical Library of the University of Manchester

Three hundred years ago amputation was one of the few operations which were performed with a technique that had been at all standardised. A very good description of the usual technique of those days is included in a remarkable book published in London in 1617 with the title " The Surgeon's Mate, or Military and Domestique Surgery discovering faithfully and plainly ye method and order of ye Surgeon's Chest, ye uses of the instruments, the vertues and operations of ye medicines, with ye exact cures of wounds made by gunshott and otherwise as namely Wounds, Apostumes, Ulcers, Fistulas, Fractures, Dislocations with ye most easy and safest ways of Amputation or Dismembring, the cures of the Scurvey, of ye fluxes of ye belly, of ye Collicke and Iliaca Passio, of Tenasmus and Exitus Ani and of the Calenture with a Treatise on ye cure of ye Plague, published for the Service of His Majesty and of the com:wealth by John Woodall."

FIG. 1

1545. Amputation is apparently being performed through the knee joint but otherwise the standard technique is used. (From Ryff, 1545.)

This is one of the great books of British surgery. It deals entirely with diseases and injuries of seamen, for " women on long voyages are rare creatures." The advice offered is as thoroughly practical as one would expect from an author who was successively Army surgeon, Surgeon-General to the East India Company, Surgeon to St Bartholomew's Hospital (1616), Naval surgeon (1627) and Master of the Barber Surgeons' Company (1633).

A few extracts from the second, enlarged edition of 1639 (with some modification of the spelling) well describe a seventeenth century amputation with several precepts that are not out-of-place to-day. " Amputation or Dismembring is the most lamentable part of chirurgery, it were therefore the honour of a Surgeon never to use dismembring at all if it were possible for him to heale all he undertaketh, but necessitie hath no law . . . Since therefore it is of necessary use, let the discreet Surgeon be ever prepared for it and to that end let the Dismembring saw be alwaies in readinesse, well fitted and cleane kept in oyly clowts to save it from rust . . . If you be constrained to use your saw let first your Patient be well informed of the eminent danger of death by the use thereof, prescribe him no certaintie of life and let the work be done with his owne free will and request and not otherwise. Let him prepare his Soule as a ready sacrifice to the Lord by earnest prayers craving mercie and helpe unfainedly: and forget thou not also thy dutie in that kinde to crave mercie and helpe from the Almightie and that heartily. For it is no small presumption to Dismember the Image of God. This done have thy other instruments ready namely a good Dismembring Knife, a

FIG. 2

1724. An upper limb amputation being performed. The technique is similar to that employed for the lower limb. (From Heister, 1724.)

small incision knife, two great square stitching needles armed with very strong thred waxed and one needle also and thred the ordinary sort to sow rowlers, likewise have ready long clowts, lesser clowts, plegents of tow greater and smaller, dorssels and buttons of tow, three broad strong rowlers or foure, of foure yards long each, with also a forme convenient for to place the Patient on, with a large boule and some ashes therein to receive the blood . . .

" This done and ready place the Patient on the mentioned forme with one strong man set behind him and another to stand before him bestriding his thigh close to his body, compassing strongly with both his hands the member which is to be taken off and holding it exceeding fast some two fingers above the place where you intend to take it away, and let another hold up his foot . . . the sharp instruments being as neere you as you can, ever hidden from the eyes of the Patient . . . Then take your dismembring knife and with a steddy hand and good speed cut off flesh, sinews and all to the bone round about the member, which done take a smaller incision knife and divide the panicle called the periosteum from the bone.

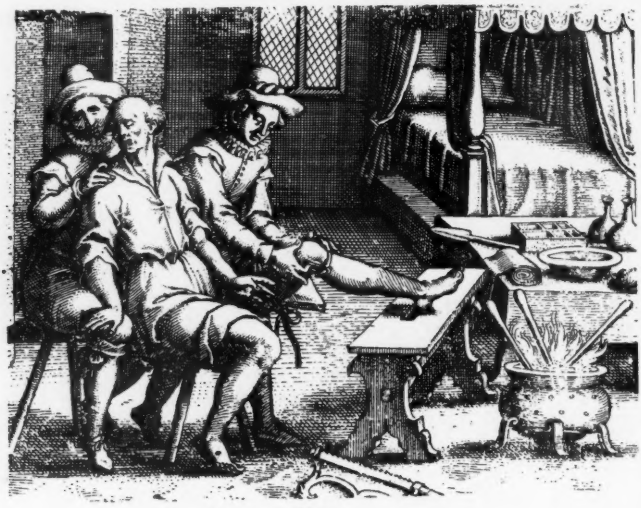


FIG. 3

1646. Wilhelm Fabry (Fabricius Hildanus) was credited wrongly with the introduction of the principle of amputation through healthy tissue above any level of demarcation. The main interest in this scene, from his collected works, lies in the preparation of cautery irons; otherwise the technique shows no difference from that described by Woodall.

It is a tough thin skin, covering all the bones of the body; also thrust your said incision knife betwixt the fossels or bones cutting away whatsoever is to bee found there with like expedition: the partie that holdeth the upper part of the legge with all his strength, griping the member together to keep in the spirits and bloud. It is also very good for the said party holding the member and flesh and sinews being cut asunder, should immediately draw or strip upward the flesh so much as he could, keeping his hold that thereby the Saw may come so much the nearer, which would occasion a quicker and better healing, the flesh being thereby made longer than the end of the bone then . . . take the two strong square needle and threads and presently after the member is taken away stitch the skin thorow on the one side and just over on the other side and with the other needle doe likewise as it were crosse over the member the other way and draw the said threds so close as you think convenient the better to stop and choake the great veines and arteries, then tye them fast and presently put buttons on to the heads of the veins and arteries then apply the restrictive plegets spread with the strong restrictive."

Finally the dressings are applied with a last instruction: "then if you will draw on a Swine's bladder which is no evil course." "The work of dismembring is best done in the morning, doe it not willingly the signe being in the place, neither the day of the full Moone, never take off any member in the joynt."

Woodall's text is not altogether original. It leans heavily on that written by William Clowes in 1596 and it is to Clowes that Woodall had dedicated "The Surgeon's Mate." The book is illustrated with fine engravings of the instruments contained in the Surgeon's Chest, but as there are no pictures of the instruments in use we have had to seek these from other sources.

FIG. 4

From a caricature by Rowlandson (1793). Although this is a much later publication, it shows, despite obvious burlesque, that the procedure presents no essential differences from the surgery of 1545. This is one of Thomas Rowlandson's best known medical cartoons and is a very personal caricature of the more eminent doctors of his day.

REFERENCES

CLOWES, W. (1596): A Profitable and Necessarie Booke of Observations. London: E. Bollifant for T. Dawson.
FABRICIUS, G. (1646): Opera. Frankfurt-on-Main: J. Beyer.
HEISTER, L. (1724): Chirurgie. Nürnberg.
POYNTER, F. N. L. (1948): Selected Writings of William Clowes. London: Harvey & Blythe, Ltd.
RYFF, W. H. (1545): Die Gross Chirurgei. Frankfurt-on-Main: C. Egenolph.
WOODALL, J. (1639): The Surgeon's Mate. London: N. Bourne.

PROCEEDINGS AND REPORTS OF UNIVERSITIES, COLLEGES, COUNCILS AND ASSOCIATIONS

GREAT BRITAIN

BRITISH ORTHOPAEDIC ASSOCIATION—SPRING MEETING, 1949

The Spring Meeting of the British Association was held in Nottingham on April 22 and 23. Papers were presented in the Great Hall of the University. The clinical meeting was held at Harlow Wood Orthopaedic Hospital where the president, Mr S. A. S. Malkin, and his colleagues from Nottingham, Mansfield, Grimsby and Derby demonstrated over one hundred cases of particular interest, including for example: bilateral Volkmann's ischaemic contracture secondary to haemophilia, myositis ossificans progressiva, dysplasia epiphysealis multiplex, and familial osteopetrosis; non-union after ischio-femoral arthrodesis treated by secondary grafting; recurrent deformity after apparently successful grafting of congenital pseudarthrosis of the tibia; traumatic spondylolisthesis treated by open reduction with unlocking of the facets; congenital spondylolisthesis with sciatic pain treated by removal of the affected disc and posterior grafting; idiopathic and paralytic scoliosis treated by grafting; a group of excellently documented cases of tendon injuries and paralysis of the hand; and a number of peripheral nerve injuries demonstrating the results of nerve suture and tendon transplantation.

The five Canadian and ten American Travelling Fellows in Orthopaedic Surgery.

R. Gariepy, H. Smith, J. J. Fahey, B. Obletz, C. Larsen, V. Inman, D. W. Blanche, W. H. Bickel, J. Leo Walker, F. P. Patterson, W. B. McKinnon, L. R. Straub (Watson-Jones, Osmond-Clarke, W. Law), B. Fowler, F. P. Dewar, J. H. Allan.

Stimulus, both scientific and social, was gained from the five Canadian and ten American travelling fellows in orthopaedic surgery who attended the meeting—F. P. Dewar (Toronto), R. Gariepy (Montreal), W. B. McKinnon (Winnipeg), F. P. Patterson (Vancouver) and J. A. Leo Walker (Montreal); and J. H. Allan (Philadelphia), W. H. Bickel (Rochester), D. W. Blanche (Los Angeles), J. J. Fahey (Chicago), S. B. Flower (Nashville), V. T. Inman (San Francisco), C. B. Larsen (Boston), B. E. Obletz (Buffalo), Hugh Smith (Memphis) and L. R. Straub (New York). They contributed to the scientific discussions, made brilliant speeches at the dinner, and founded the A.B.C. Club by which to promote interchange between the orthopaedic services of America, Britain and Canada. Other visitors included Professor J. Delchef and Dr A. C. Steenebruggen, from Belgium.

Function of the clavicle—*Dr Verne T. Inman* (San Francisco, California) illustrated experiments, including the insertion of transfixion pins into his own clavicle and acromion, and those of his colleagues and students, which demonstrated that elevation of the upper limb involved rotation of the clavicle about its long axis, and that fixation of the clavicle to the coracoid process prevented elevation above shoulder level. In eight patients the clavicle had been excised completely without loss of function except that extra effort was needed to hold objects above the head. The operation was suggested as an adjunct to arthrodesis of the shoulder joint.

Anomalies of innervation of the hand muscles—*Mr Tom Rowntree* (London) reported investigation of 688 median and ulnar nerve lesions treated at the Wingfield-Morris Orthopaedic Hospital, Oxford, and the Institute of Orthopaedics, Royal National Orthopaedic Hospital, London. Only cases with proved axonal division of the nerves had been accepted—102 median nerve lesions and 124 ulnar nerve lesions—the observations being checked in some cases by procaine block of the unaffected nerves. The innervation described in text-books was found in 33 per cent. of cases—the median nerve supplying abductor pollicis brevis, opponens and flexor pollicis brevis, and the ulnar nerve supplying adductor pollicis, the hypothenar muscles and all the interossei. The flexor pollicis brevis had a purely ulnar supply in 32 per cent.; it had a dual supply from both median and ulnar nerves in 16 per cent. There was an anomalous supply in 20 per cent. including some with innervation of all hand muscles by the median nerve, and others with innervation of all muscles by the ulnar nerve. In a few cases procaine nerve block had demonstrated anastomosis between the ulnar and median nerves in the forearm. These results emphasized the fallacy of diagnosis of nerve lesions on the evidence of muscle power alone.

Extensor apparatus of the digits—*Dr S. Benjamin Fowler* (Nashville, Tennessee) said that the extensor communis tendons had no useful insertion into the proximal phalanges; and the inability of these tendons to extend the interphalangeal joints of the claw hand depended upon hyperextension of the metacarpo-phalangeal joints. If such hyperextension was prevented by tightness of the joint capsules the inter-phalangeal joints could be extended actively by the long extensor tendons. The use of tendon transplants of extensor pollicis brevis, extensor indicis proprius and extensor digiti quinti, to prevent or overcome metacarpo-phalangeal hyperextension, and thus promote function after paralysis of the intrinsic muscles, was illustrated.

Tendon surgery in the hand—*Mr R. G. Pulvertaft* (Derby), in a masterly presentation, showed some remarkable results of difficult tendon sutures and demonstrated shortening of the dorsal expansion in old cases of mallet finger, repair of boutonnière lesions of the extensor expansions, tendon transplantation of extensor indicis proprius for rupture of extensor pollicis longus, suture of divided flexor tendons of the fingers, and replacement of the flexor tendons by tendon grafts.

Rupture of extensor pollicis longus tendon after Colles' fracture—*Mr David Trevor* (London) described five cases of spontaneous rupture of the extensor pollicis longus tendon, which he attributed to aseptic necrosis from ischaemia, that had been treated by simple bridging of the gap with No. 6 Nylon sutures. The sutures were removed three months or more after operation. Good union had occurred, and photographs of excellent results were shown at intervals ranging up to four years after operation. *Mr B. L. McFarland* (Liverpool) suggested that the ischaemic changes might be caused by strangulation of the tendon in the pulley opposite Lister's tubercle. *Mr R. G. Pulvertaft* (Derby) said that he preferred to transplant the tendon of extensor indicis proprius into the distal part of the ruptured tendon.

Pronation fractures of the forearm with special reference to the anterior Monteggia fracture—*Mr E. Mervyn Evans* (Birmingham) presented clinical and experimental evidence that the anterior Monteggia fracture-dislocation was produced by forced pronation and longitudinal compression. In full pronation, the radius and ulna crossed in contact; and if pronation force continued beyond the normal range of the movement the ulna fractured near its middle and prized the radial head forwards out of the joint. Closed reduction by supination, without internal fixation, had succeeded in nine of eleven cases. *Mr F. W. Holdsworth* (Sheffield) confirmed that " compounding " of this fracture was always forwards. In children he treated this injury by conservative measures but in older people he usually plated the ulna. Excision of the radial head gave bad results. *Sir Reginald Watson-Jones* (London) agreed that the rotatory force of pronation might be responsible when there was a spiral fracture of the ulna but doubted whether this was the mechanism when the fracture was transverse. One form of this injury was clearly due to direct injury—the " baby-car " fracture or, as the Americans more picturesquely put it, " the side-swipe " fracture. He believed that internal fixation of the ulna by plate or intramedullary nail was often necessary in adults but that open reduction of the dislocated radial head was not usually advisable. *Mr W. Sayle-Creer* (Manchester) doubted whether supination alone would maintain reduction which was sometimes prevented by the torn capsule.

Fractures of the neck of the femur—*Mr P. C. Elmes* (Oxford) reported a statistical analysis of 115 fractures of the femoral neck treated by closed nailing. The clinical results were good in thirty-five cases, fair in forty-two, and bad in thirty-eight. Anatomical results were estimated in sixty-one cases examined radiographically more than two years after operation. Sound bony union had occurred in thirty-nine

cases but there was ischaemic necrosis with collapse of the femoral head in nine of these. Factors which significantly affected the incidence of ischaemic necrosis were sex, site of fracture, interval between injury and operation, interval between operation and weight-bearing, and accuracy of reduction.

Trochanteric fractures of the femur—*Mr R. C. Murray* (Inverness) reported one hundred trochanteric fractures of the femur treated conservatively by simple weight-traction with a medial rotation bandage, early movement being encouraged. He presented strong evidence that, with good nursing care, the results of conservative treatment were better than the results of operative internal fixation with nails and blade plates. The mortality was lower and the functional results were equally good. *Mr William Gissane* (Birmingham) referred to thirty-two patients with trochanteric fractures treated by the Capener-Neufeld nail of whom thirty-one had regained excellent movement in the hip and knee joints. *Mr K. H. Pridie* (Bristol) supported conservative treatment.

Ischio-femoral arthrodesis of the hip—*Mr W. B. Foley* (Oxford) gave a cinematographic demonstration of arthrodesis of the hip-joint of the Brittain type by posterior open approach through a Kocher incision as described by Trumble. Plaster of Paris was applied with the patient still prone.

Lumbo-sacral fusion by metallic fixation and grafts—*Dr L. R. Straub* (New York) discussed lumbo-sacral fusion in eighty cases treated by the method described in 1943 by Dr Philip Wilson in which metallic fixation was combined with the Hibb's operation, supplemented by bone chips, both autogenous and from the " bank." One patient died of post-operative shock. Of the others, sixty-nine showed radiographic evidence of sound bone fusion at the end of six months, the evidence being accepted only when several films had been taken in different positions of strain. Failure in five had been at the lumbo-sacral joint, and in five at a higher level. Recently, metallic fixation had been combined with a cortical graft, and twenty-five of twenty-seven patients had shown solid fusion. Among 103 patients treated without metallic fixation, there had been three deaths and twenty-nine failures.

Arthrodesis of the ankle by lateral approach and fibular graft—*Dr J. A. Leo Walker* (Montreal), with the aid of most beautiful cinematography and cartoons that might have been devised by Walt Disney, demonstrated the technique of this operation in which fusion of the joint was supplemented by fixation of the split fibula to the lateral aspect of the tibia and talus.

Radiological diagnosis of recent lesions of the lateral ligament of the ankle—*Mr J. Roland Hughes* (Preston) said that radiographic examination of the ankle joint in the position of full inversion as described by Watson-Jones was now well-established. He had examined ninety injured ankles and ninety normal ankles. About 6 degrees of tilt represented the transition from normal to abnormal. The lateral ligament was lax when the ankle joint was plantar-flexed, and tight when it was dorsiflexed. In the cadaver, important tilting did not occur until there had been division of both the anterior talo-fibular ligament and the capsule as far as the lower limit of the articular surface of the talus. It was then possible to tilt the talus through 30 degrees. These ligaments were torn by a movement of plantar-flexion combined with rotation about the medial malleolus. If tilting occurred through more than 18 or 20 degrees there must be rupture not only of the anterior talo-fibular ligament but also of the calcaneo-fibular ligament and the intervening capsule. *Mr J. Grant Bonnin* (London) considered that tilting up to 15 degrees indicated rupture of the anterior talo-fibular ligament; tilting of 15 to 30 degrees, rupture of both anterior talo-fibular and calcaneo-fibular ligaments; and tilting of more than 30 degrees, rupture of all three parts of the ligament. It was important to X-ray the talus of the uninjured foot which could be tilted about 4 degrees in 10 to 15 per cent. of cases.

Osteochondritis dissecans of the elbow joint—*Mr Norman W. Roberts* (Liverpool) said that osteochondritis dissecans of the elbow joint was almost confined to the male. It was commonest under the age of fifteen years; and it affected both elbows in about one-quarter of the cases. The lesion was usually in the capitellum but sometimes in the radial head. There was often diffuse cystic change; detached loose bodies were usually multiple. The radial head was enlarged and premature epiphysial fusion might occur. Association with preceding injury was difficult to establish. When there were loose bodies in the elbow joint radiographs often revealed flattening of the capitellum and pronounced enlargement of the radial head, thus indicating that the source of the loose bodies was unsuspected osteochondritis dissecans rather than osteoarthritis, which was a complication rather than a cause. *Mr H. Jackson Burrows* (London) said that bone in the separated fragment distinguished osteochondritis dissecans from chondromalacia which was often mistaken for it. Osteochondritis dissecans in the elbow joint presented a more complicated picture than the corresponding lesion in the knee. It might be that the knee-joint lesion was less simple than had been supposed—or alternatively that the elbow change represented a different and more complicated pathology, possibly akin to osteochondritis juvenilis of the metatarsal head (Freiberg's infraction). *Mr J. Rowland Hughes* (Preston) compared enlargement of the radial head to coxa magna and thought that it might be caused by laxity of the capsule secondary to the changes in the capitellum.

Nerve-root pain of vertebral origin not dependent on disc prolapse—*Professor J. Delchef* (Brussels) emphasized that apophysial sprain was a real entity and that arthritis of the posterior articulations might be an important cause of sciatica of single root origin. *Mr J. R. Armstrong* (London) recalled that the first sacral root, the most frequently affected, was unrelated to a posterior joint; and furthermore that sciatica was not usually relieved by arthrodesis. He considered that almost all cases were due to intervertebral disc lesions.

Election of Fellows and Members—Elections to fellowship and membership of the Association were made at the Executive meeting:—

Corresponding Member	P. G. K. Bentzon, Denmark	*Associate Members*	H. H. Kennedy, Chesterfield
			A. W. L. Kessel, London
Fellows . . .	F. C. Durbin, Exeter		H. Keith Lucas, Liverpool
	J. H. Mayer, Tunbridge Wells		T. H. Norton, Grimsby
	K. Stanger, Newcastle on Tyne		W. A. J. Pike, Auckland, N.Z.
			C. G. Rob, Thirsk
Associate Members .	J. K. Cunninghame, Wellington, N.Z.		F. H. Stevenson, Stanmore
	Johnstone Dickie, Leicester		T. E. Stoker, Cheam
	P. G. Essex-Lopresti, Birmingham		G. S. Storrs, Bedford
	James Garden, Carluke		G. J. Walley, Windsor
	R. N. Grant, Truro		R. I. Wilson, Exeter

Clinical Research Sub-committee—At the first meeting of the Clinical Research Sub-committee on April 22, 1949, it was agreed that policy should be based on these considerations:

1) Experimental research was essentially local, and hardly came within the purview of the committee, but much could be done to promote collective clinical research.

2) The suitability of subjects for collective research should be determined on the basis of controversial interest, rarity, or a combination of both.

3) Any fellow or member who had particular interest in a subject for clinical research should submit his proposal to the committee, if possible with the names of those who might collaborate with him.

4) If the proposal was supported, a working group of fellows and members would be formed, and a chairman selected, the group arranging to meet regularly on the day preceding each meeting of the Association.

5) The group should agree on a uniform scheme of documentation and on such variations in investigation or treatment as might be desirable; and submit their proposals to the Clinical Research Sub-committee.

6) Requests for financial aid would be transmitted to the Executive of the Association.

Clinical subjects were considered and four working groups approved:

Traumatic dislocation of the hip joint—Chairman, E. A. Nicoll.
Slipped upper femoral epiphysis—Chairman, H. Jackson Burrows.
Calvé's osteochondritis of the spine—Chairman, B. E. McFarland.
Cup arthroplasty of the hip joint—Chairman, H. A. Law.

ROYAL COLLEGE OF SURGEONS OF ENGLAND

Award of the Honorary F.R.C.S. to Professor R. I. Harris (Toronto)—No honour awarded by the Royal College of Surgeons has been acclaimed more generally than that of the jealously guarded honorary fellowship conferred on Dr R. I. Harris who was twice wounded and twice decorated in the 1914–18 War, served as Consultant to the Canadian forces in the recent war, is one of the acknowledged leaders of Canadian Surgery, Professor of Orthopaedic Surgery in the University of Toronto, and recent president of the American Orthopaedic Association. He took an important part in establishing British-American co-operation in the publication of this Journal and in promoting the travelling fellowship of young orthopaedic surgeons from Britain, Canada and America. It was fitting that conferment of the honorary fellowship by the President of the College, Lord Webb-Johnson, should have been witnessed by the five Canadian and ten American surgeons who are the most recent travellers, as well as by the thirteen British travellers of last year. The audience filled the College to overflowing. No sooner had Dr Harris been admitted than he accepted the duty to which every Fellow aspires—the delivery of a Hunterian Lecture.

Hunterian Lecture on Spondylolisthesis—*Professor Harris* said that spondylolisthesis was of special interest among the causes of low back pain because of its problems of etiology, diagnosis and treatment. The essential defect in the neural arch between the superior and inferior articular facets was most common in the fifth lumbar vertebra. The defect led to instability of the spine and allowed the body of the affected

vertebra to slip forward, carrying with it the superimposed spinal column, while the small posterior fragment maintained its normal relationship to the subjacent vertebra. When the defect was present without displacement the condition was termed spondylolysis. In such cases detection was difficult without oblique radiographs of the spine.

Little was known of the cause. The defect was said to occur in 5 per cent. of adult cadavers but was not found once in 200 foetuses recently examined, and these observations did not support the view that the defect was developmental. On the other hand there was little definite evidence to suggest that it resulted from injury, either at birth or in later life. Despite much work it had to be admitted that the origin ot the defect was unknown.

Discussing the clinical aspects, Professor Harris said that the two main features were pain and deformity. Both arose from instability of the spine. Pain was of two types: 1) lumbar pain, due to instability alone, aggravated by weight-bearing and activity, and relieved by rest; 2) pain from root pressure, due either to associated prolapse of the lumbo-sacral disc with pressure on the first sacral root, or to narrowing of the intervertebral foramina with compression of the fifth lumbar roots. When root pain was caused by prolapse of a disc it was usually unilateral; but when caused by narrowing of the intervertebral foramina it was bilateral. Deformity was produced partly by anterior displacement of the body of the affected vertebra with the spinal column above it, and partly by secondary compensatory changes —increased lumbar lordosis and backward rotation of the pelvis, which brought the sacrum more nearly vertical.

Careful judgment was required in the choice of treatment, which might be conservative by means of a spinal support, or operative by spinal fusion. In general, fusion was recommended for young patients and those with severe symptoms. Occasionally, pre-operative correction of severe deformity was attempted. As to the type of operative fusion, the transabdominal method was not recommended. The anterior graft had to cross the relatively avascular area of the intervertebral disc; and the approach did not permit exploration for possible causes of root pressure. Posterior fusion was satisfactory if the graft was massive and its anchorage secure. The graft should not extend higher than the vertebra above the lesion; and adequate post-operative recumbency should be maintained for at least four months. In his own clinic, twin tibial grafts had been used, supplemented with cancellous chips. The lower end of each graft was slotted into a window cut in the back of the sacrum; above, the grafts were anchored to the spinous process by stainless steel wire. In a series of sixty-seven patients treated by operation, the result was satisfactory in fifty-six.

ROBERT JONES DINING CLUB

On April 29, 1949, the evening of the day that Professor R. I. Harris was elected an Honorary Fellow of the Royal College of Surgeons, a meeting of the Robert Jones Dining Club was held. Membership of this club is limited to the number of those who, on the occasion of Robert Jones' seventieth birthday, contributed to the Birthday Volume, together with a few distinguished overseas members. Dr Harris was the guest of honour and was elected a member.

BRITISH MEDICAL ASSOCIATION—ANNUAL MEETING

ORTHOPAEDIC SECTION, JUNE 1949

At the annual meeting of the British Medical Association, held in June 1949 at Harrogate, Mr Reginald Broomhead of Leeds presided over the Orthopaedic Section.

Structure and functions of the synovial membranes—*Professor D. V. Davies* (London) described the synovial membrane as the least differentiated joint structure. In the embryo it was differentiated from intersomal mesoderm and was continuous with the cartilage which later formed the bone ends. From the beginning, the joint capsule was continuous with the fibrous periosteum. The peripheral part of the intersomal mesoderm soon became vascularised and the central area then became a cavity. Embryological continuity of the synovial membrane with chondrogenous and osteogenous tissue explained the occasional formation of cartilaginous and bony loose bodies in the synovial membrane. The lining cells, appropriately called mesothelial cells, had been referred to as synovial fibroblasts; they could be replaced by the underlying cells but stained differently and showed characteristic reaction to irritation—multiplying, enlarging and becoming several layers thick, just as did peritoneal cells. They seldom became free in the joint fluid. They were the main source of synovial mucin, though not staining characteristically with mucicarmine. They behaved differently from fibroblasts in tissue culture, secreting a mucinous substance and a proteolytic enzyme which liquefied the fibrin medium. They took an active part in removing particulate matter from the joint cavity and in haemarthrosis and villonodular synovitis were often found to be loaded with haemosiderin. They probably also removed protein by lysis, as seen after transient arthritis. The synovial villi, which were scanty in man and increased in number with age, were often avascular and their

function was uncertain. The synovial membrane contained reticulo-endothelial elements, able to take up trypan blue or gold especially when inflamed. The blood vessels were superficial and contributed to the circulus vasculosus which penetrated and nourished the peripheral part of the articular cartilage. The nerves, which had been studied by Gardner, were few; they followed the vessels and had no elaborate ending. Little was known of the development or function of the fat pads that were characteristic of synovial joints.

Varieties of pathological reactions in human synovial tissues—*Dr C. H. Collins* (Leeds) had examined and studied two hundred and fifty biopsy or autopsy specimens of synovial membrane and recorded the reactions as: changes in the synovial cells; proliferative and exudative phenomena; and leucocytic reactions. The synovial cells had three peculiar properties: the production of extracellular mucin; capacity to form a serosal lining; and ability to change into phagocytic histiocytes. Malignant synoviomata illustrated the tendency of synovial cells to elaborate mucin and form serosal membranes; and phagocytic properties were seen in haemosiderin phagocytosis. Giant cells, from fusion of histiocytes, were found in many conditions including rheumatoid arthritis. Haemorrhagic and oedematous reaction took place readily and gave an exaggerated aspect to inflammatory changes. Perivascular lymphatic infiltration was massive only in chronic inflammatory conditions like tuberculous and rheumatoid arthritis. Lymphocytic aggregates and plasma cells were often found in rheumatoid arthritis. Polymorphs were present in small numbers in all chronic inflammatory conditions and entered the joint fluid more readily than lymphocytes. All these changes resolved quickly. *Mr D. Engel* (Dewsbury) had perfused joints in animals after intravenous injection of acid and alkaline dyes. None except the acid dyes with small molecules reached the joint cavity, and they did so in proportion to their diffusibility. The amount was increased in acute inflammation and reduced in chronic inflammation or after lumbar sympathectomy.

Denervation of the hip joint in osteoarthritis—*Mr H. Petty* (Leeds) described innervation of the hip joint and said that complete denervation had not been attempted though the danger of producing a neuropathic joint was more theoretical than real. Success in relieving osteoarthritic pain had been variable. He had followed the practice of Tavernier in resecting the obturator nerve and the nerve to quadratus femoris in nineteen patients. Three were completely relieved of pain; twelve still suffered intermittent aching; two had more serious pain but were improved and believed that the operation had been worth while; two were unimproved. It is to be noted that the duration of follow-up averaged no more than three to nine months. *Mr H. Jackson Burrows* (London) recalled that the simplest denervation of the hip joint was by capsulectomy as practised many years ago by the late R. C. Elmslie. It was most easily done through Kocher's incision.

Mould arthroplasty of the hip joint—*Mr R. Broomhead* (Leeds) reported one hundred arthroplasties of the hip joint for congenital dislocation, rheumatoid arthritis, ankylosing spondylitis, osteochondritis juvenilis, slipped epiphysis with late osteoarthritis, malum coxae senilis, infective arthritis and injury. The operation had been performed in patients up to the age of seventy years. Technical difficulties included shortness of the femoral neck which might call for trochanteric transplantation, and flexion and lateral rotation deformities which tended to persist after operation. The results depended not so much on the joint as on the condition of the muscles which was imperfect in old cases of rheumatoid arthritis and ankylosing spondylitis. Patients with osteoarthritis due to osteochondritis juvenilis or slipped upper femoral epiphysis regained at least 50 per cent. of the normal range of movement and could work all day without pain. Young adults with osteoarthritis from other causes regained only about 20 per cent. of normal movement; but patients with malum coxae senilis, the largest group, obtained a good return of all movements except rotation. Co-operation of the patient in after-treatment was imperative; the fat woman and the deaf patient presented difficulties. The most troublesome complication was new bone formation in the field of operation. Two deaths had occurred, both shortly after operation. Mr Broomhead said that secondary operations—so-called revisions—were disappointing. The importance of meticulous after-care, with the use of crutches for six months after operation, was emphasized.

The role of surgery in rheumatism—*Mr Petty* discussed the treatment of patients with acute rheumatoid arthritis. He showed many examples of mould arthroplasty of the hip joint, and a most excellent result of bilateral arthroplasty of the knee joints.

Physical treatment of arthritis—*Dr H. F. Turney* (London) reviewed the physical treatment of arthritis of the hip and knee joints by radiant heat and diathermy, and active exercises with Guthrie-Smith slings, roller skates and the deep pool. Pistany packs were just mud. Faradism had a place secondary to active exercise. Diet played no part save in weight-reduction. Intra-articular injection of lactic acid with procaine might be useful but procaine alone, and even simple saline injection, was no less effective. *Mr Grant Waugh* (Sunderland) considered that the acidity of the fluid that was injected was important. Procaine solutions were usually prepared from the hydrochloride and were acid. Procaine had often to be discontinued because the patient became sensitised.

Rheumatology in the United States of America—*Dr G. D. Kersley* (Bath) recorded observations made at the Seventh International Congress on Rheumatic Diseases in New York and discussed the histochemistry of collagenous and other connective tissues, the histology of rheumatoid arthritis, and the hereditary factor in ankylosing spondylitis. The subject of most interest was the use of cortisone and ACTH in rheumatoid arthritis. Cortisone was related to adrenal cortical hormones concerned with electrolyte and water metabolism; and ACTH was an anterior pituitary hormone. The dramatic improvement gained on administration of these hormones was equalled by dramatic deterioration on their withdrawal. Cortisone had been effective not only in rheumatoid arthritis but also in rheumatic fever, gout, ankylosing spondylitis, scleroderma, dermato-myositis and generalised lupus erythematosus.

Upper limb pain from lesions of the thoracic inlet—*Professor Lambert Rogers* (Cardiff) said that neural pain, tingling in type and mostly on the ulnar side of the limb, was caused by pressure on the lower trunk of the brachial plexus by a cervical rib or fibrous band, the sharp tendinous anterior margin of the scalenus medius, or an abnormal scalenus anterior. Vascular pain had the aching character of ischaemia and was often associated with cutaneous colour changes. It was caused by compression of the subclavian artery between the clavicle and a cervical rib or abnormal first rib. The differential diagnosis was from writer's cramp—a neurosis, and irritation of a cervical root by rupture of a cervical disc at the C.5, 6, 7 level of which radiological evidence might be found. Thirty-three operations had been performed, twenty-five for the scalenus syndrome and eight for costo-clavicular compression. *Mr A. C. Bingold* (Leeds) said that the differential diagnosis should also include Pancost's syndrome, caused by compression of the brachial plexus and cervical sympathetic by a tumour at the thoracic inlet with severe pain on the inner side of the limb, wasting of the intrinsic muscles of the hand, Horner's syndrome, venous congestion in the arm and neck, and a radiographic shadow at the inlet. *Mr H. M. Hill* (Western Australia) recalled that radiographic evidence of an old cervical intervertebral disc injury did not necessarily imply that the symptoms were related to it.

Closed fractures of the shafts of the radius and ulna—*Mr E. Mervyn Evans* (Birmingham) said that in fractures of both bones of the forearm rotation deformities were often left unreduced. The key to orientation of the proximal radial fragment was the radiographic appearance of the bicipital tuberosity. This varied not only with the position of the bone but in different individuals; but it was always the same on the two sides of the same individual if the forearms were similarly rotated. Antero-posterior radiographs were taken of the normal forearm in positions of 90 degrees, 120 degrees, 150 degrees and 180 degrees of pronation. An antero-posterior radiograph of the fractured forearm was then taken and the appearance of the bicipital tubercle compared with the various normal films. The distal radial fragment was orientated in correspondence with the known position of the proximal fragment by manual traction against the resistance of a sling, looped over the upper arm, and continued for at least ten minutes. Simple plaster of Paris fixation was used. Of thirty-five closed fractures with displacement treated conservatively, twenty-seven showed less than 30 degrees limitation of movement. One needed bone-grafting of the radius for non-union and the others united in an average period of eleven weeks. *Mr F. W. Holdsworth* (Sheffield) agreed that rotation deformity had been neglected. Difficulties often arose from interposed soft tissues and from the obliquity or comminution of the fracture and consequently he had adopted open reduction and plating despite the dangers of non-union and infection. He had had three cases of infection among eighty-five such fractures. After operation no external fixation was used. *Mr I. Lawson Dick* (Bradford) agreed that, notwithstanding the dangers of plating, there were some fractures that demanded it, notably those of the lower part of the radius with radio-ulnar subluxation. *Mr H. H. Langston* (Southampton) endorsed the importance of rotation deformity but urged that open operation should not be used routinely. *Mr N. Ross Smith* (Bournemouth) considered that even when open reduction was required, internal fixation, with its risk of delayed union, should be avoided if the fragments could be interlocked. *Mr G. A. Pollock* (Edinburgh) agreed that internal fixation should be avoided. *Dr F. W. Hanford* (Northern Rhodesia) considered that the risk of infection in open reduction had been underestimated. *Dr H. M. Hill* (Perth, Western Australia) agreed that plates were often necessary; their use might be supplemented by grafting with iliac bone to accelerate union.

ROYAL SOCIETY OF MEDICINE

SECTION OF ORTHOPAEDICS—1949

Practical value of peripheral nerve repair—*Mr H. J. Seddon* gave his presidential address on February 1, 1949. He pointed out that, apart from Stopford's data published after the first World War, there were few accurate accounts of the end-results of nerve suture. Opinions ranged from uncritical optimism as to the success of primary nerve suture to profound pessimism as to the failure of secondary repair of the sciatic and ulnar nerves. The five Nerve Injury Centres set up in Great Britain at the beginning of the second World War had achieved remarkable uniformity in documentation and the Medical Research Council now had records of a very large series of cases traced for periods of three to five years.

The proportion of good recoveries increased steadily up to three years after suture; but there was little change between the third and fifth years. The figures were encouraging and selected cases were demonstrated to show what was meant by good recovery: one man could climb telegraph poles after sciatic nerve suture; another played squash; one was able to ski after suture of the lateral popliteal nerve.

There were three unavoidable causes of failure. 1) Recovery was usually imperfect when the nerve lesion was so proximal as in injuries of the brachial plexus. Except in the case of lesions of the upper trunk, little if anything was to be gained from operative repair at this level. 2) Associated injuries to blood vessels, muscles, tendons and joints sometimes precluded useful recovery, though it was remarkable how many limbs formerly considered to be damaged beyond repair could be made useful. 3) Some lesions, such as traction injuries and ischaemic damage of main nerve trunks, were too extensive for repair by any means at present available.

The avoidable causes of failure were more numerous. Stiffness of joints and shortening of muscles should be avoided at all costs. The remedies were simple: preservation of mobility by careful passive movements; limitation of splinting to that which was needed to prevent overstretching of muscles; and control of muscle atrophy by electrical stimulation.

Delay in repair of a severed nerve was harmful because: 1) Schwann cells, which were responsible for the formation of a cellular bridge between the sutured stumps, reached a peak of activity some few weeks after division and thereafter lost their power of proliferation; 2) shrinkage of Schwann tubes caused by Wallerian degeneration in the peripheral stump became irreversible, so that outgrowing axons were unable to attain the diameter necessary for their proper function; 3) the motor end-plates atrophied and disappeared, and after late suture were usually replaced by inferior structures; 4) atrophy and fibrosis of denervated muscle was progressive and ultimately irreversible; 5) sooner or later the peripheral stump shrank to such a degree that accurate apposition became impossible.

It was of course desirable that a nerve should be repaired with the least possible delay but, even in favourable circumstances, primary suture had not always given satisfactory results. Zachary and Holmes had compared a series of cases treated by primary suture with similar cases dealt with by early secondary repair. There were many satisfactory results after primary repair but, as a whole, the results were far superior in cases treated by early secondary repair. It was often found that even wounds that appeared to be cleanly incised caused severe intraneural changes which became evident later as zones of fibrosis. Sometimes the suture was technically unsatisfactory because of the poor state of the epineurium. In other cases separation of the stumps resulted from dragging at the suture line when the flexed joint was extended. All these hazards could be avoided by the delayed operation, at which it was possible to mobilise the nerve adequately and place the line of suture in favourable surroundings.

When complete paralysis was associated with an open wound the policy should be to explore the nerve within a few weeks of healing of the wound because complete division was found in over half the cases. Resection was indicated if there was unmistakable evidence of intraneural fibrosis in an undivided nerve, or if there was any but the most trivial incomplete division.

Imperfect recovery was sometimes due to inadequate mobilisation of the stumps, especially after wounds at the wrist. In half these cases mobilisation as high as the elbow was required. Conversely, over-ambitious attempts to close large gaps by mobilisation and acute flexion of joints gave poor results. There was a biological limit to the length of gap that could be closed with safety and, when very large gaps had been closed, later straightening of the limb had inflicted serious traction lesions. There was a critical length of resection for every nerve and a gap approaching or exceeding this was better closed by autogenous grafting which had now been proved reliable. Inadequate resection of the stumps was an uncommon cause of failure, but mistakes were occasionally made in taking too little from the peripheral stump which might suffer ischaemic change, particularly after wounds of great violence. There might also be failure due to post-operative separation at the suture line by a haematoma. This could be detected by radiography after attaching to the stumps, close to the suture line, opaque, inert markers—small gold plates.

In closed fractures with nerve injury there was division of the nerve trunk in only one case in five; in all others good recovery occurred spontaneously. Exploration should therefore be limited to those cases in which signs of recovery failed to appear in the time required for axons to grow from the site of damage to the most proximal of the paralysed muscles.

Amputation surgery and limb-fitting—The Summer Meeting of the Section of Orthopaedic Surgery was held on June 25, 1949, at Queen Mary's Hospital, Roehampton—the main centre in Great Britain for amputation surgery and limb-fitting. The morning was occupied by clinical demonstrations of the preparation of stumps for prosthesis, the methods used in limb fitting, the new suction socket for the lower limb, and improved types of upper limb prosthesis. *Mr L. Gillis* (London) emphasized the importance of co-operation between experienced surgeons and limb makers. Vast experience gained in the Ministry of Pensions had proved invaluable in determining the length and characteristics of the perfect stump and surgeons should accept the recommendations of limb-fitting specialists. A careful technique was essential.

All bleeding vessels should be ligated meticulously. Coagulation by diathermy should be avoided. The deep fascia should be sutured to ensure freedom from adhesion between skin and deep tissues. In children, epiphysiodesis was useful in preventing disproportionate growth of bone and soft tissues. The problem of painful phantom limb remained unsolved.

Painful stumps—*Dr Dimant* said that pain might arise from skin, scar tissue, muscle or bone; or from the effects of vascular impairment. An analysis of 600 cases of painful stump was presented.

The fitting of short stumps—*Dr McKenzie* discussed difficulties in fitting short thigh stumps. Function was more natural with a well-fitted above-knee prosthesis than with a tilting-table. In the past it had seldom been possible to fit a stump-controlled prosthesis with less than six inches, but a stump as short as four and a half inches could now be fitted satisfactorily.

Traumatic skin loss below the knee—*Mr R. Battle* discussed skin replacement by thin Thiersch grafts, dermatome grafts, local flaps, cross-leg flaps and tubed pedicle grafts. A free Thiersch graft was often satisfactory even when applied directly over bone; if it proved unstable it could be replaced later by a pedicled graft. Local flaps were potentially dangerous, particularly in unskilled hands. The length of the flap should never be greater than twice its width. Cross-leg flaps were convenient because the proximity of donor and recipient areas allowed direct transfer. Tubed pedicle grafts were often preferred because of the healthy and supple character of the transferred skin. The usual technique was to carry the skin on the wrist from abdomen to leg.

UNIVERSITY OF OXFORD

NUFFIELD PROFESSOR OF ORTHOPAEDIC SURGERY

Josep Trueta, who has been elected Nuffield Professor of Orthopaedic Surgery in the University of Oxford, was born, a Catalan, in Barcelona on October 28, 1897, son of Raphael Trueta, physician in Barcelona, and great-grandson of Antony Trueta, surgeon of the Army of General Lancaster in the war between Spain and France in 1795. In 1929 he was appointed chief surgeon of the " Caja de Provision y Socorro," an organisation treating 40,000 accidents a year. In 1933 he was elected assistant professor of surgical pathology in the University of Barcelona and in 1935 became chief surgeon to the " Hospital de la Santa Creu i Sant Pau," Barcelona.

Josep Trueta of Barcelona who has been appointed Nuffield Professor of Orthopaedic Surgery in the University of Oxford.

In 1936 the Spanish Civil War began. Trueta's position as chief surgeon of the largest hospital in Catalonia, and one of the largest accident services in Spain, gave him the opportunity of developing the method of treatment of war injuries that was later used extensively in the treatment of casualties in the Allied Armies. On February 27, 1939, he reached this country at the invitation of surgeons who had seen his work in Barcelona; and in September he was invited to join the staff of the Wingfield-Morris Orthopaedic Hospital.

His publications, which are too extensive to be listed here, comprise thirty-two papers on civil orthopaedic surgery, twenty on the surgery of war, and six contributions to the literature of crush syndrome and the renal circulation. He is also author or joint author of six books on the surgery of peace and war.

In 1940 he was elected an Honorary Fellow of the British Orthopaedic Association and in 1943 was awarded the D.Sc. Honoris Causa by the University of Oxford. In 1947 he gave the Balfour Lecture in the University of Toronto, and the Annual Lecture on Surgery at the Royal College of Physicians and Surgeons of Canada. He was elected an Honorary Fellow of the Royal College of Surgeons of Canada. In 1948 he gave the Carmalt Lecture, Yale, and the Friesner Lecture, Mount Sinai Hospital, New York; and he was presented with the Prix Laborie, with special medal, of the French Acadamie de Chirurgie.

Trueta's name has been associated particularly with the closed plaster treatment of wounds. It is perhaps not fully realised that, while credit is due to Winnett-Orr for the closed plaster treatment of osteomyelitis whether of haematogenous origin or associated with compound fractures, Trueta's practice differed in that his closed plaster was part of the initial treatment of gunshot wounds and other open injuries, and that he insisted that the plaster should be applied only after complete excision of all contaminated, devitalised and ischaemic tissues. Trueta emphasized the importance of rest of soft tissues as well as of bones, particularly in relation to tissue infection. In organising the reception of air-raid casualties he had learnt that the interposition of a first-aid post between the " incident " and the hospital gave rise to delay that was harmful, and was needless in all but minor and superficial wounds.

Trueta, who was a professor of surgery and leader of the profession in his own country, came to England at the request of British surgeons and offered the fruits of his unique experience in the Spanish Civil War. He has played a valued part in all the clinical activities of the hospital in Oxford. His appointment as Nuffield Professor of Orthopaedic Surgery will add distinction to the Wingfield-Morris Orthopaedic Hospital and the Oxford Medical School.

REGIONAL ORTHOPAEDIC MEETINGS IN GREAT BRITAIN

SOUTH-WEST ORTHOPAEDIC CLUB

A meeting of the South-West Orthopaedic Club was held in Truro on May 14, 1949, at the Royal Infirmary under the chairmanship of Mr J. H. Hood.

Congenital club foot—Unselected cases of talipes equino-varus were shown at all stages of treatment. The treatment favoured was manipulation and fixation with strapping. Points of interest that emerged in discussion were: 1) despite good initial correction, relapse, with inversion of the heel, was liable to occur at about the age of eighteen months; 2) the calf muscles showed hardening and atrophy at the same age; 3) associated torsion of the tibia appeared to be more common than with the Denis Browne method of treatment.

Spina bifida with meningocoele—*Miss Forrester-Browne* pleaded for conservative treatment of spina bifida. The skin usually broke down because of the contact of moist " nappies." By means of a perspex shield, shaped like a tunnel and open at both ends, the skin could be protected and kept dry. The meningocoele then shrank to proportions not requiring surgery.

Dupuytren's contracture—*Mr Rentoul* discussed the operative treatment of Dupuytren's contracture. A longitudinal incision was used, regardless of skin creases, and the thickened fascia together with its phalangeal prolongations was excised. No splintage was used. Six cases with excellent results were demonstrated. It was claimed that a longitudinal incision did not cause contracture or keloid change and that it facilitated the technique of operation and reduced the likelihood of sloughing. There was a lively discussion on the merits of subcutaneous tenotomy.

Other discussions—*Miss May* discussed the treatment of pneumococcal arthritis of the knee joint by chemotherapy. *Mr Price* demonstrated Calvé's disease of the spine with regeneration of the affected vertebra. *Mr R. A. Grant* outlined the organisation for the treatment of injuries in Cornwall. *Mr E. J. Kirwan* discussed the manipulative and operative treatment of Monteggia fracture-dislocations. *Dr F. Wilson*, geriatrician for the Cornwall area, outlined plans for the rehabilitation of old patients with chronic polyarthritis.

INSTITUTE OF ORTHOPAEDICS

ROYAL NATIONAL ORTHOPAEDIC HOSPITAL, LONDON

Visitors' week—A group of European surgeons who visited the Institute of Orthopaedics in February 1949 included: Drs Benassy, Descamps, Meary, Radadier and Vigneau from Paris; Dr Nievergelt from Zurich; Dr Sibay from Leyden; Drs Vereecken and Verjans from Ghent; Drs Coquelet and de Marneffe from Brussels; Dr Steenebruggen from Liège; Dr Parisel-Leclercq from Ostend; Dr van de Voorde from Antwerp; and Dr Jacobson from Oslo. The treatment of peripheral nerve injuries was reviewed by Mr H. J. Seddon and Mr D. M. Brooks. The technique of cup-arthroplasty of the hip joint was discussed by Mr J. I. P. James. The technical details of clinical photography of orthopaedic cases was presented by Mr Whittey.

At the country branch of the hospital modern methods of treatment of poliomyelitis were demonstrated in the gymnasium and swimming pool. Mr J. T. Scales demonstrated the use of plastic materials for orthopaedic splintage. The standard treatment of tuberculosis was presented by Mr E. J. Nangle. Clinical cases were shown by Mr K. I. Nissen and Mr J. A. Cholmeley. Methods of plastic surgery were demonstrated by Mr Matthews.

One day was spent at Luton where Mr L. W. Plewes and Dr Anderson showed the organisation of an accident service at the Luton and Dunstable Hospital and the rehabilitation service that has been established in the Vauxhall Motor Works. Another day was devoted to the study of handicapped children at the School for Physically Defective Children, Willesden, where Miss Pritchard demonstrated the efforts of achondroplasics, patients with muscular dystrophy, and other crippled children, in their group drill to music. The Cripples' College of the Royal National Orthopaedic Hospital and its associated workshops were visited under the guidance of Colonel Churchill. On the last day a visit was paid to Stoke Mandeville where Dr Guttmann showed recent advances in the early treatment and late rehabilitation of patients with traumatic paraplegia.

It is hoped that this visitors' week, which began with a welcoming speech in French by Mr H. J. Seddon, and finished with a visit to Twickenham where France and England met in a rugby football match, will be the forerunner of similar visitors' weeks in future years.

Lecture by Professor Gruca—Dr Gruca, professor of orthopaedic surgery in the University of Warsaw, gave a lecture at the Institute of Orthopaedics, in February 1949, on Excision of the Hip with Osteotomy for the Restoration of Movement after Tuberculous Infection.

ROBERT JONES AND AGNES HUNT ORTHOPAEDIC HOSPITAL

In recent numbers of the Journal it was recorded that much of the Robert Jones and Agnes Hunt Orthopaedic Hospital had been destroyed by fire. Subscriptions began to pour in within a few days. Safe in the confidence of the support that this hospital commands from many thousands of former patients and voluntary workers, in this and many other countries, rebuilding was begun almost at once. If any had doubted the wisdom of such confidence their doubts have now been resolved. The subscription list totals £41,000. The spirit of voluntary effort is not dead.

MEMORIAL TO DAME AGNES HUNT

It was one of the ideals of Agnes Hunt that cripples should be relieved from the hardships of institutional life and be allowed to enjoy the comforts and privacy of a normal home; and as a memorial to her it is proposed to create a village for disabled persons in the region of the Robert Jones and Agnes Hunt Orthopaedic Hospital and the Derwen Cripples' Training College. The village will consist of bungalows, some to accommodate one or two individuals and others to house the family of crippled parents. It will include community and recreational facilities. The cost of each bungalow will be in the region of £1500 to £2000 and they will be named in accordance with the wishes of the donors.

The cost of rebuilding the hospital was met almost without appeal—but this is a new demand and an appeal is now being made to all those who believe that the memory of Agnes Hunt should be perpetuated.

PORTLAND TRAINING COLLEGE FOR THE DISABLED—HARLOW WOOD

Princess Elizabeth laid the foundation stone of the Portland Training College for the Disabled, Harlow Wood, Nottinghamshire, on June 29, 1949. Describing the objectives of the College, which will be associated closely with the Harlow Wood Orthopaedic Hospital, Mr S. A. S. Malkin said that it was through the initiative of a few citizens of Nottingham, led by the Duchess of Portland, that the orthopaedic hospital came into being in 1929. Since then it had grown and prospered, and was rendering an important service to the district. But no matter how great the advances of medical science, there would always be some who, on account of the severity or the nature of their disabilities, would never return to their former occupations. To them the future seemed black. Nevertheless, with special training, most of them could be made fit for new work suited to their abilities. They would become self-supporting, independent and happy members of the community. It was to give this training and opportunity that the Portland Training College was being built. The College would serve mainly the four counties of Nottinghamshire, Derbyshire, Lincolnshire and Leicestershire. It would be established on a voluntary basis, outside the sphere of the National Health Service.

MINERS' WELFARE COMMISSION

REHABILITATION OF INJURED MINERS

In the annual report of the Miners' Welfare Commission for 1948 which was published recently, the consulting surgeon, Mr E. A. Nicoll, reviewed the growth of the present rehabilitation service. He recalled that ten years ago the word " rehabilitation " was practically unknown in the medical world; yet its value was already recognised by a few pioneers. As early as 1935 a rehabilitation clinic had been established in the Lanarkshire coalfield by Alexander Miller of Glasgow. The importance of reinstatement of injured men in the working community was beginning to be recognised, but at that time the machinery for co-ordinating rehabilitation and social service was lacking. In 1939, Berry Hill Hall, near Mansfield, was opened as a rehabilitation centre with one surgeon and one physiotherapist, and with accommodation for fifteen patients. Although the Centre was started as an experimental project its value was quickly recognised by the coal owners who agreed not only to make it permanent but to extend it to accommodate forty patients. In 1942, the Ministry of Fuel and Power requested the Miners' Welfare Commission to provide similar facilities throughout Great Britain. A medical advisory committee was appointed under the chairmanship of Sir Reginald Watson-Jones, with Mr E. A. Nicoll as consulting surgeon. To-day there are eight centres serving all the major coalfields, and three associated schemes serving the minor coalfields of Cumberland, Kent and North Staffordshire.

The results of the year's work are impressive. No less than 92·3 per cent. of injured patients treated in the centres were able to return to useful and economic work in the pits, and of these 63 per cent. resumed their full pre-accident work. These figures represent the results of the more difficult cases occurring in the mining area—many lesser injuries do not require the elaborate facilities for treatment and social service that have been built up at the centres.

In the clinical field the extensive accummulation of well-documented material for analysis and review will form a valuable contribution to clinical research. Already small teams of surgeons are concentrating on the investigation of clinical problems that are so uncommon that it would take one surgeon, working alone, almost a lifetime to solve them.

AUSTRALIA

AUSTRALIAN ORTHOPAEDIC ASSOCIATION

Membership of the Australian Orthopaedic Association now includes forty active members, twelve associate members, three honorary members and ten corresponding members. The 1949 annual meeting was held in Sydney from June 1 to 4. The main subjects for discussion were: Surgical tuberculosis by *Mr D. W. L. Parker*; Treatment of fractures about the elbow joint by *Mr W. R. Gayton*; Treatment of slipped epiphysis of the femur by *Mr E. F. West*; and The role of tendon transplantation in restoration of function after paralysis by *Mr H. Crawford*.

The Association welcomes the formation of a Postgraduate Education Committee by the Royal College of Surgeons of England and will advise regarding Australian applicants for post-graduate orthopaedic training in Great Britain.

INDIA

SOCIETY FOR THE REHABILITATION OF DISABLED AND CRIPPLED CHILDREN

The Society was envisaged in 1947 by a group of voluntary workers and became a registered society in October 1948. Its objects are: to organise hospitals and clinics for the diagnosis, care and treatment of disabled and crippled children; to educate public opinion on the problem of such afflicted children; and to collect statistics relating to it. Although the work of the Society has so far been restricted to the Bombay area it is hoped that it may ultimately be expanded to deal with the problem of crippled children throughout India. The finding of suitable premises has been a major difficulty. At present the work is being carried on in a military hutment, converted for use as an out-patient physiotherapy and rehabilitation clinic. The clinic is staffed by ex-army physiotherapeutic technicians under the direction of a panel of doctors. Dr M. G. Kini, M.C., M.Ch. (Orth.) has been appointed orthopaedic surgeon to the society.

Up to the end of 1948, 233 patients had been accepted for treatment. Most were cases of poliomyelitis. There is no distinction as to caste, creed, sect, nationality or class. Transport to the clinic is provided. Rapid expansion of the activities of the society is foreseen. Within the next two years it is proposed: to establish a children's orthopaedic hospital on the most modern lines; to organise a teaching institution for the supply of trained physiotherapists; to establish an orthopaedic workshop; to found a school for crippled children; and to set up an institute for research into the problems of poliomyelitis.

CZECHOSLOVAKIA

CZECHOSLOVAK SOCIETY OF ORTHOPAEDIC SURGERY

The annual meeting of the Czechoslovak Society of Orthopaedic Surgery will be held in Prague on September 24–25, 1949. The subjects for discussion will include the general treatment of fractures, and osteoarthritis.

GREECE

PRESENT STATE OF ORTHOPAEDIC SURGERY IN GREECE

Mr St J. Buxton (London) was invited recently to make a visit to Greece with the purpose of surveying and reporting upon the present state of orthopaedic surgery in that country and the main problems that are being encountered in its development.

War surgery—Surgeons working in the army, particularly at the base hospitals in Athens and Salonika, had had great experience of military surgery and the standard of operative work was high. Surgery at the forward area also appeared to be performed competently as judged by the low incidence of infection in wounds and compound fractures evacuated to base hospitals. The value of infrequent dressings of limb wounds was not fully recognised and it was still a common practice to cut windows in the plaster for daily dressing. The treatment of peripheral nerve injuries did not include all modern methods of investigation, and tendon transplantation at the wrist was often carried out before the radial nerve had been explored with a view to suture.

Amputations—It had been estimated that about 2500 amputations were performed between 1945–48, and that the total number of amputees was about 4000. Ten per cent. of amputations were of the upper limb. The standard levels of amputation were known and practised, but many unhealed granulating stumps resulting from the guillotine type of operation were seen. Limb-fitting showed some lack of co-operation between the operating surgeon and the limb factory.

Civilian orthopaedic surgery and the care of crippled children—Although the treatment of injuries was under the care of general surgeons the speciality of orthopaedic surgery was recognised and there were a number of experienced surgeons who had travelled to study the methods used in other countries. Methods of investigation, radiography and operative treatment were of a high standard. Surgical tuberculosis was prevalent. There was an excellent pavilion type hospital at Voula. Immobilisation of tuberculous joints was much less strict than in Britain. Streptomycin was used when abscesses were present.

Rehabilitation and physiotherapy were not yet fully developed in their application to war surgery, civilian accident work, or the treatment of long term orthopaedic cases.

GERMANY

The German Orthopaedic Congress—The Orthopaedic Congress was held in Munich on April 21–23 under the presidency of *Professor G. Hohmann.* Congenital dislocation of the hip joint was discussed by Professor Lanz (Munich), Professor Scaglietti (Florence), Dr Rohlederer (Esslingen), Professor Pitzen (Münster), Dr Niederecker (Würzburg), Dr Detzel (Munich), Professor Schede (Sandersbusch), Professor Lindemann (Hanover), Dr Hepp (Hamburg), Professor Hackenbroch (Cologne) and Professor Storck (Giessen). The discussion continued throughout the whole of the first day and covered the pathology of preluxation and subluxation, the indications for early preventive treatment, the management of contractures and late stiffness, the problems of torsion and coxa valga luxans, and treatment by plastic reconstruction of the acetabular roof and rotation osteotomy. On the second day deformities of the hands and fingers were discussed by Professor Weil (Heidelberg) and Dr Witt (Bad Tölz). Rehabilitation and training of the disabled was discussed and, at a visit to Bad Tölz, demonstrations were given by Professor Max Lange.

MEXICO

Mexican Society of Orthopaedic Surgery—The Mexican Society of Orthopaedic Surgery was established in 1944 and its activities are being extended. *Dr Luis García Figueroa* is president of the Board and the secretary is *Dr Ladislao Solares.* The Society includes sixteen charter members, three active members and two honorary foreign members. Correspondence is welcomed: the Secretary's address is Sinalao 71, Mexico, D.F.

CORRESPONDENCE

Polyostotic Fibrous Dysplasia—Albright's Syndrome—*Mr Charles K. Warrick* (Newcastle upon Tyne) writes to point out that in his paper on Polyostotic Fibrous Dysplasia, published in this Journal in May 1949 (volume 31-B, No. 2, page 180) the height of the patient, Case 2, J. M., was recorded as 5 feet 11 inches but should have been 5 feet 1 inch.

Book Reviews

ESSENTIALS OF ORTHOPAEDICS. By Philip WILES, M.S., F.R.C.S., F.A.C.S., Hon. Orthopaedic Surgeon, Middlesex Hospital and King Edward Memorial Hospital, Consulting Orthopaedic Surgeon, Royal Surrey County Hospital. Formerly: Brigadier, A.M.S.; Consulting Surgeon (Orthopaedics), Middle East Force, and Persia and Iraq Force; and Consulting Surgeon, Eastern Command, India, and 12th Army, S.E.A.C. 10×7 in. Pp. xv+486, with 365 text-figures and 7 coloured plates. Index. 1949. London: J. & A. Churchill, Ltd. Price 42/–.

Mr Philip Wiles has addressed himself to the task of expounding the essentials of orthopaedics for the benefit of the undergraduate student, the surgical resident and the general practitioner. It is unlikely that any two senior clinical teachers will agree on what is "essential" in any field of medicine or surgery. Every monograph is bound to reflect the writer's perspective view of the situation which confronts him. But, in actual fact, it is the personal "slant" which makes this attractively written work of Mr Wiles so appropriate for his chosen audience. The book opens with an excellent chapter on postural defects—a subject which the author made his own some years ago. This is followed by a chapter on back pain, full of practical common sense. The reader is thus introduced to the core of clinical orthopaedics at the outset. This approach deserves the highest praise. Later sections are conventionally devoted to pyogenic infections, tuberculosis of bone and joint, chronic arthritis, tumours of bone, other bone diseases and diseases of the central nervous system. Apart from these selective chapters, orthopaedic disabilities are described in their anatomical relationship to the joints of the trunk and the upper and lower limbs. It would be easy to criticise the relative space allocated to particular topics: for instance, twenty pages on bone tumours, with a tendency to over-emphasis on rarities (e.g., Fig. 327), and only the same number of pages for the chapter on chronic arthritis, in which it must be admitted Mr Wiles has managed to condense the essential features of modern orthopaedic practice. There are also some notable omissions, doubtless in the cause of brevity. A chapter on the principles of fracture treatment—not an easy subject to present in synoptic fashion—would have been valuable; and we miss the historical approach and bibliography of "classical" references, so important for the intellectual stimulus of the undergraduate and house surgeon. The work is bound to take its place amongst the popular student text-books; future editions will undoubtedly be called for; and Mr Wiles will be tempted to expand his admirable book. The selection of essentials will not be easy, but by reason of his ripe experience and sound judgment he is well fitted to undertake it.
—Harry PLATT.

FIG. 35
Two methods of manipulating the dorsal spine.

SOURCE BOOK OF ORTHOPAEDICS. By Edgar M. Bick, M.A., M.D., F.A.C.S., Dipl. Orth. Surg., Associate Orthopaedic Surgeon, The Mount Sinai Hospital, New York; one time Regional Consultant Orthopaedic Surgeon (Army), European Theatre of Operation. Second edition. 9½×7 in. Pp. xii+540, with 31 figures. Index. 1948. Baltimore: The Williams & Wilkins Company. Price $8.00 (44/-).

The arrival of the second edition of Dr Bick's admirable source book has been long awaited. It is doubtful whether this book is well enough known, particularly in Britain, and it is to be hoped that this new edition will give the work the very wide recognition which it deserves, for it is unique, up to date, authoritative and very interesting. It is far more than its title implies. Besides representing a commendably complete and very painstaking collection of the sources and original papers upon which modern orthopaedic surgery is based, it is an interesting and exciting account of the development of our art and science. It should be read for pleasure and for profit by all who are interested in the historical background of our craft, and should form part of the compulsory reading of anyone who thinks that he has devised a new operation, a new technique or a new theory.

Revision and recasting have been very thorough. The first part of the book (which deals with orthopaedic surgery before 1900) has been revised, but is substantially what it was. Of the five new pages which have been added to this section most are taken up with new illustrations. The second part of the book, however (on contemporary orthopaedic surgery and its recent sources), has been nearly doubled in length and has been brought well up to date. There are, for example, admirable surveys of the surgery of the Second World War, of the modern views of the physiology of bone and of their development, and even of such subjects as genetics as applied to orthopaedic surgery. There is a useful, if admittedly incomplete, list of orthopaedic journals and an interesting but necessarily brief account of the development of orthopaedic hospitals. The many excellent features of this book make its one grave fault—a series of irritating mistakes probably due to poor proof-reading—particularly regrettable; the more so because some have remained uncorrected since the first edition. "Colins Mackenzie" (p. 10), "Filkin of Northwick" (p. 76), "Abernathy" (p. 82), "Intercranial haemorrhage" (p. 4), a mistake in the sex of Miss Honor Fell (pp. 94–95), "Fairbanks" for Sir H. A. T. Fairbank (p. 167), "is" for "his" (p. 208), and "Coffee" for Coffey (p. 211) are among the jars which upset the reader. One must also criticise Dr Bick's rather ready acceptance of oft-repeated surgical legends which are probably quite untrue (such as the idea of the introduction of the ligature by Paré and of amputation above the line of demarcation by Fabry), and of some stories which are certainly false (such as the statement on p. 79 that Pott sustained a Pott's fracture, whereas the injury which he did sustain in 1756 was a compound fracture higher up the leg). The greatness of Sherrington's contribution to orthopaedic surgery, albeit an indirect one, is not clearly realised; and a British reader may be forgiven for pointing out that the surgical history of the Second World War did not begin at Pearl Harbour. These are, however, remediable faults, for this is a fine book, stressing once again that "what is considered 'modern' is but the sum total of previous observations re-classified, re-interpreted . . . and occasionally multiplied," a lesson which none of us can rehearse too often.—D. Ll. Griffiths.

THE BRITISH JOURNAL OF SURGERY. War Surgery Supplement No. 2. WOUNDS OF THE EXTREMITIES. Five reviews collected and edited by H. J. Seddon, D.M., F.R.C.S. 10×7½ in. Pp. x+271–374, with 74 figures, 4 in colour. 1949. Bristol: John Wright & Sons, Ltd. Price 12/6.

The time has now come when the surgery of the last war, although still fresh in mind, can be seen in proper perspective, and the editors of the British Journal of Surgery are to be congratulated on producing the series of War Surgery Supplements of which this volume is the second. It is essential that progress made during the war is accurately and concisely recorded, and, of the several methods available, the one chosen here, a series of reviews by representative surgeons, is undoubtedly the best. Mr Seddon has been happy in his choice of contributors because they have not only given an accurate account, but they have also managed to convey the sense of urgency, peculiar to war surgery, that arises from the pressure of a seemingly unending stream of casualties piling up at the front door.

The outstanding improvement in the medical services in this war lay in the administrative appreciation that no surgeon can do his work properly unless the patient reaches him quickly and in as good condition as possible, unless he has modern means of resuscitation at hand, and unless he has good lines of communication behind him. F. A. R. Stammers describes in the opening article how these things were achieved in the Italian Campaign. He follows the wounded man until his arrival at a base hospital, and deals with first aid, resuscitation, chemotherapy, splintage, transport, the general principles of primary surgical treatment and the modifications that may be necessary to suit varying local conditions. It is the most authoritative and succinct review of this subject yet published, and it might well be learnt by heart both by those planning the Army Medical Services of the future and the surgeons enrolled in it. Stammers is perhaps over-modest about the part he played, together with his fellow-consultant Harold Edwards, in planning the medical

organisation of the Italian campaign, for it was they who arranged the first large-scale experiment in delayed primary suture which proved so successful that it rapidly superseded the routine use of closed plaster methods. Ronald Furlong and J. M. P. Clarke contribute a valuable account of the treatment at base hospitals of missile wounds involving bone. They discuss first the general principles concerned and then special difficulties encountered at selected sites. Their experience is particularly instructive because they received their patients direct from the forward surgical units and were able to hold most of them for definitive treatment. J. C. Scott records the remarkable change in the prognosis of penetrating wounds involving joints that has come from improved methods of splinting during transport, and from chemotherapy. Even at the time of the North African Campaign in 1942 the mortality and the amputation rates in wounds of the knee had both been reduced to one quarter of the 1917 figure, and by 1945 they were negligible. In spite of the remarkable results achieved by New Zealand surgeons, and later by many others, with very early mobilisation of missile wounds of the elbow, Scott still prefers to fix the elbow until healing is complete. H. J. Seddon's knowledge of peripheral nerve injuries is unrivalled, and no more worthy pen could be employed to present the long-awaited results of the experiences of this war. One cannot help marvelling —as he describes in succession non-operative treatment, the indications for primary and secondary operative repair, nerve-grafting, and, finally, the end-results of suture—at the immensity of the painstaking labour continued day after day for years that has made this record possible. The end-results are assessed on the basis of the objective criteria elaborated by the Nerve Injuries Committee of the Medical Research Council, and he now makes available, for the first time, the really reliable information that is essential if the surgeon is to reach a rational decision as to his conduct in a particular case. J. J. Mason Brown, in the final review, deals with injuries to peripheral arteries in an equally authoritative and comprehensive way. He has not such a happy rôle to fill, because surgical progress in this field has been less striking; he has to record that the results of primary ligation of main arteries are still unsatisfactory, and that arterial restoration by grafting remains uncertain in spite of many brilliant attempts and a few outstanding successes.—Philip Wiles.

A PRACTICE OF ORTHOPAEDIC SURGERY. By T. P. McMurray, C.B.E., M.B., M.Ch., F.R.C.S., (Edin.). Professor of Orthopaedic Surgery, Liverpool University, Honorary Orthopaedic Surgeon, David Lewis Northern Hospital, Director of Orthopaedics, Royal Liverpool Children's Hospital. Third edition. $8\frac{1}{2} \times 6$ in. Pp. viii + 444, with 191 figures. Index. 1949. London: Edward Arnold & Co. Price 30/–.

It is scarcely fitting to carp about a book that is twenty-one years old and has survived six printings; yet to the reviewer this book is showing its age. The principles of orthopaedics may not change—and, as might be expected from a disciple of Robert Jones, they are here clearly stated—nevertheless treatment changes, sometimes because of increased knowledge and sometimes at the whim of fashion. Dare one say that the present edition has not followed these changes closely? Royle's operation, Whitman's reconstruction of the hip, and enucleation of the tarsal bones for club foot, might be omitted. The chapter on acute osteomyelitis needs complete reconstruction because of the discovery of the antibiotics. In the treatment of infantile paralysis the shift of emphasis from rigid splintage has not been stressed. Since in orthopaedics early diagnosis is so important, it is a pity that tuberculosis of the spine is illustrated by a child with gross kyphosis: tuberculosis of the spine will not be discovered early until the practitioner is accustomed to notice the small break in the uniform curve of a child's spine in the bending position. Nor will a slipped epiphysis of the upper end of the femur be picked up early if he expects a history of injury. It is strange to find from Liverpool operation recommended as the only treatment for a ruptured intervertebral disc. Certain ommissions strike one: references to senile osteoporosis of the spine and stenosing tendovaginitis would be welcome. Claw toes might well be considered apart from pes cavus.

Although the production, we are told, is of war economy standard, it could hardly be better. Most of the radiographic reproductions are so good that an occasional obscure picture is noticeable: the picture of spondylolisthesis is nothing but a blur. Publishers should realise that bad illustrations are worse than useless. This book is such an old favourite of this reviewer that he would wish the next edition to be perfect. —George Perkins.

TUBERCULOSIS OF THE KNEE JOINT: AN EXPOSITION WITH SPECIAL REFERENCE TO COURSE AND TREATMENT BASED ON STUDIES OF THE LITERATURE AND ON AN ANALYSIS OF A MATERIAL OF PATIENTS OBSERVED THROUGH MANY YEARS. By Johannes Mortens. Translated from the Danish by Axel Andersen. $10 \times 6\frac{3}{4}$ in. Pp. xiv + 550, with 6 figures, 13 tables and 243 charts. 1948. Copenhagen: Einar Munksgaard. London: H. K. Lewis & Co., Ltd. Paper cover. Price 37/6.

Dr Mortens describes tuberculosis of the knee as it occurred in 118 patients treated at the coastal hospital at Refnaes, Denmark, and in the surgical tuberculosis department of the Finsen Institute, to whose chief, Johannes Meyer, he pays tribute. These were patients in whom the diagnosis was not in doubt.

He analyses the results of conservative and operative treatment in the light of follow-up examinations performed more than ten years later. He compares these results with comparable results by other surgeons, to obtain which he has submitted the literature on conservative treatment and operative treatment to exhaustive analysis. He also gives a valuable history of the treatment of tuberculosis of the knee. Thus, much of the work consists of a mass of detail: and it becomes a little difficult to see the wood for the trees. But answers to certain questions emerge, and these are so important and are so well authenticated that Dr Mortens's effort is amply rewarded.

The questions that Dr Mortens sets out to answer are whether conservative treatment of the disease in children gives good results or whether excision is preferable in some. If so, what are the risks of shortening or deformity? About adults he discusses whether excision should be performed early or late. By conservative treatment he means bed rest in an open-air hospital, splintage (which seems to be less rigidly applied than here) and actinotherapy for which the Finsen Institute is famous and which is used to the full. Sixty-two children received conservative treatment only: twenty recovered with a good range of movement and little damage to the joint; seven others had a range of movement varying from 40 degrees up to 140 degrees; thirteen got bony ankylosis; seventeen developed fibrous ankylosis; three patients died; and in two the disease did not heal. Of fifteen children who had resection after conservative treatment fourteen developed bony ankylosis; three children who had primary resection did badly. Dr Mortens goes on to discuss resection in childhood and finds that bony ankylosis can usually be obtained though it takes longer to develop than in the adult. The lowest age limit set for excision is nine years. Notable shortening did not often occur in cases in which there had been little destruction of cartilage or erosion of bone, but he found that it was common after excision of joints that had suffered gross destructive change. Yet these are the joints for which excision may particularly be required; but there is less risk of shortening if excision is performed when the lesion is healing. Dr Mortens would favour also excision if the disease is still active six or seven years after the onset. He considers deformity after excision to be a more formidable complication than shortening. To avoid deformity the limb must be fixed in full extension at operation. Many patients must wear a splint for three or four years after operation, and others must have a tenotomy of the flexor tendons. It is clear that Dr Mortens has no enthusiasm for the treatment of tuberculosis of the knee in children by excision, and from his research he finds no encouragement to depart from conservative treatment as a routine measure. For adults, on the other hand, conservative treatment is recommended only if it does not interfere with their wage-earning capacity; excision is the method of treatment recommended if the general condition of the patient is satisfactory and the disease in the joint not very active.

In the chapter on pathogenesis Dr Mortens retains the distinction between a primarily osseous and primarily synovial type of lesion, but admits that the disease is present in both bone and synovial membrane as soon as it becomes recognisable. The joint lesion is regarded as part of a haematogenous infection, in which tubercle bacilli are deposited in the latent form in various parts of the body, and the development of overt lesions is governed by circumstances that determine tissue resistance. Associated pulmonary tuberculosis was especially common.

There is much else in this book that repays study. There are questions to ask Dr Mortens: for example, whether constitutional treatment can safely be curtailed in adults, in whom the incidence of associated lesions is so high; and whether the prognosis for associated pulmonary tuberculosis is improved by operation into a tuberculous joint. There can be no doubt about the merit of this outstanding contribution to the literature of skeletal tuberculosis.—M. C. WILKINSON.

OSTEOPATHIE RARE. By C. CASUCCIO. Libero docente di Clinica Ortopedica nell'Universita di Bologna. Primo aiuto dell'Istituto Ortopedico Rizzoli. Prefazione di F. Delitala. 10 × 7½ in. Pp. xii + 545, with 292 figures. Index. 1949. Bologna: Edizioni Scientifiche Instituto Rizzoli. Price 5500 Lira.

This exhaustive study of the rarer diseases of bone, excluding infections and neoplasms, is based on the vast material provided by the Institute Rizzoli and a very wide study of the relevant literature in all languages. Like Ludin, Casuccio classifies the diseases as either atrophic or hypertrophic and sclerosing. This method has considerable disadvantages, but none will be satisfactory until more is known of the etiology and pathology of these conditions. There is some lack of balance in the amount of space given to the description of various diseases. For example, the epiphysial changes in cretins receive but brief mention, while eosinophil granuloma occupies nineteen pages, excellently illustrated and documented. Again, some of the chapters contain a conglomeration of conditions which, in this country, might not all be considered closely related. The radiographs and histological plates are excellent and together with the description present a clear picture of the conditions and what is known of their etiology, pathology, course and treatment. This book is a mine of information and provides knowledge that could otherwise be obtained only with great difficulty from original sources in many languages. The style is clear and the Italian intelligible to the reader with the slightest knowledge of the language. The bibliography is comprehensive,

excellent, and exceptional in that the full title of the original article is mentioned. This is a valuable and unique book of reference on an important but complex subject.—V. H. Ellis.

This monograph is based on a comprehensive study of the uncommon skeletal affections included in the great collection of documented material now available in the Rizzoli Institute. A wide search of the literature has also been carried out, and the ample bibliography shows that full recognition has been paid to the contributions of others on this subject. The work bears the imprint of the famous Bologna School of Orthopaedics. The author has adopted the convenient method of classifying skeletal disease under two main categories: 1) atrophic lesions (osteoporotic; osteolytic; and dystrophic); 2) hypertrophic lesions (hyperostotic; osteosclerotic; osteoplastic). Each skeletal disease is presented in clear and simple language with the clinical, radiographic and histological features described in proper perspective. The format of the volume is admirable. The author is to be commended for producing a work of reference which all orthopaedic surgeons will find of the greatest assistance.—Harry Platt.

AN ATLAS OF TRAUMATIC SURGERY. By Josep Trueta, M.D., Barcelona; D.Sc. (Hon.), Oxon.; F.R.C.S. (Hon.), Canada. Surgeon, Wingfield-Morris Orthopaedic Hospital, Oxford. 10×8 in. Pp. viii+150, with 188 figures. 1949. Oxford: Blackwell Scientific Publications, Ltd. Price 30/-.

The development of the closed plaster technique in the treatment of open injuries is closely associated with the name of Trueta. Like many other orthopaedic surgeons who were impressed by the work of Baer and of Winnett Orr in the later part of the First World War, he introduced the technique into his civilian practice, and then, in the Spanish War of 1936–38, he was able to apply it on a big scale to the treatment of war wounds. The results he achieved were outstanding, and the publication of his book "Treatment of War Wounds and Fractures" at a most opportune moment in 1939 was followed by the general adoption of the ideas he had so ably advocated. In the recent war, as a member of the surgical staff of the Wingfield-Morris Orthopaedic Hospital, Oxford, he was able to continue working along the same lines, and his present book is the record of an achievement no less remarkable than the first. "An Atlas of Traumatic Surgery" is a straightforward narrative and pictorial record of the progress and treatment of forty patients with compound fractures and large wounds of the upper and lower limbs chosen from 127 serious casualties treated since the day of the landing in Normandy. As the hospital included a peripheral nerve centre, most of the injuries were complicated by peripheral nerve injuries. No attempt is made to describe details of technique, and the text consists of unvarnished extracts from the hospital records; the reader is therefore free to form his own conclusions, and he cannot fail to be impressed by the magnificent results that were regularly achieved. The book, however, is not only a record; it is also a challenge, for the author states in his preface, "It is primarily to meet the need of these future workers [of a new generation] that this book is published, although it is hoped that it may be of interest even now, as a record of clinical experience, by providing material for comparison of results for those still facing similar problems." The difficulty of judging honestly the results in the treatment of major injuries is notorious, and it is impossible to set the performance of one surgeon against that of another because the precise conditions can never be known. It is, however, noticeable that, although treatment was carried out at a time when delayed primary and secondary suture were in common use, they were performed on a minority of the larger wounds in this series. It is also apparent that re-excision of wounds was frequently required—it may be because the primary forward surgery was for some reason below the usual standard. But even should some other surgeon claim better or quicker results, this would in no way detract from Trueta's achievement. He has done fine work and given us a magnificent permanent record of it, a record of lives saved and desperately mangled limbs restored to useful function; and any surgeon of the present or a future generation should be proud if he can do as well.—Philip Wiles.

Fig. 139

Condition of the wound and fracture on admission, twenty-five days after injury. The great muscular loss makes it very difficult to maintain the fragments in correct position.

Fig. 144

Almost two years after injury the condition of the limb is knee flexion 80 degrees. One year later is 105 degrees.

PLASTER OF PARIS TECHNIC. By Edwin O. Geckeler, M.D., Professor of Orthopaedic Surgery, and Chief of the Fracture Service, Hahnemann Medical College and Hospital, Philadelphia. Second edition. 9×6 in. Pp. xiv+220, with 236 figures. Index. 1948. London: Baillière, Tindall & Cox. Price 16/6.

This book has already been reviewed briefly in an American number of the Journal, but without critical comment. The teaching of plaster technique requires very orderly instruction. Here, however, elementary and advanced details alternate, and the numerous illustrations lack continuity. Some of the techniques described must lead to trouble or complaint. Thus, few surgeons would agree with the following captions. "If the ankle has been immobilised in equinus by mistake, a rectangular window will permit a change in the position without pinching the skin." "Short hip spica: the back view shows the buttock on the affected side completely covered with plaster." "Application of plaster jacket (two table method): no padding is necessary." "Certain conditions of the knee, as separations of the lower femoral epiphysis, require immobilisation in hyperflexion." To continue in this vein, the cast shown for a mallet finger is completely straight at the proximal joint; flat-foot deformity is treated by inversion of the whole foot; and a plaster figure-of-eight is used for fractures of the clavicle in children. In short, it may be fairly said that many of the techniques advocated show a wide variation from good contemporary practice in this country.—K. I. Nissen.

FRACTURES AND DISLOCATIONS FOR PRACTITIONERS. By Edwin O. Geckeler, M.D. Fellow of the American College of Surgeons, Fellow of the American Academy of Orthopaedic Surgeons, Fellow of the American Association for the Surgery of Trauma, Diplomate of the American Board of Orthopaedic Surgery. Fourth edition. 9½×6½ in. Pp. xii+371, with 344 figures. Index. 1948. London: Baillière, Tindall & Cox. Price 27/6.

The object of this book is to provide a simplified yet practical account of the treatment of fractures and dislocations for general practitioners and senior students. This is no easy task, and the author has approached it with a due sense of realism and responsibility. He devotes a good deal of space to fundamental principles and fundamental techniques; and, in dealing with individual fractures for which a variety of treatments has been advocated, he confines his advice to one method which in his experience has proved to be the "simplest and most reliable for the best functional result"—a most refreshing reaction from the present tendency to dispute the right of any patient to be cured without a display of surgical virtuosity. But there is also danger in over-simplification; this Dr Geckeler avoids by indicating quite clearly those conditions in which complicated and difficult techniques are indispensable. Here he is at pains to tell his readers how to handle such cases until more skilful help can be obtained. The key-note of his advice in such circumstances is: "If there is doubt as to the treatment of a difficult case, it is far better to call a consultant at once than to meddle and manipulate repeatedly."

In matters of detail there is room here and there for criticism. Volkmann's ischaemic contracture is *not* due to venous obstruction. In adults, half an inch of shortening does *not* usually cause a permanent limp. Distraction should be added to the important causes of delayed union. Total division of the spinal cord does *not* lead to a fatal issue "regardless of the best care" (there are eight paraplegia centres in the author's own country proving the exact opposite). The removal of ununited transverse processes is an operation strongly to be condemned and not easy to perform—whereas open reduction of fractures of both bones of the forearm, which the author condemns, is widely practised, with good results, even in the comparatively conservative atmosphere of Great Britain. Finally, most surgeons will be unnecessarily alarmed to read that in one out of every three dislocated hips the femoral head undergoes avascular necrosis.

The author has a clear, direct style and the text is well illustrated. A short bibliography concludes each chapter, but with few references to the literature of the last five years.—E. A. Nicoll.

SURGERY ORTHODOX AND HETERODOX. By Sir William Heneage Ogilvie, K.B.E., D.M., M.Ch., F.R.C.S., Hon. F.A.C.S., Hon. F.R.C.S. (Canada), Hon. F.R.A.C.S. Surgeon, Guy's Hospital and Royal Masonic Hospital. 9×6 in. Pp. v+241, with 30 figures. 1948. Oxford: Blackwell Scientific Publications. Price 12/6.

In this volume Ogilvie has collected a number of lectures and addresses on surgical topics. They reveal the thoughts of an elder statesman which, if not profound, could not be more delightfully expressed. The music of the words reminded the reviewer of the many occasions he has sat enchanted listening to the oratory of the author.—George Perkins.

ATLAS OF NEUROPATHOLOGY. By Wm. Blackwood, M.B., F.R.C.S.E., Assistant Pathologist, National Hospital, Queen Square; formerly Senior Lecturer in Neuropathology, Edinburgh University. T. C. Dodds, F.I.M.L.T., F.I.B.P., F.R.P.S., Laboratory Supervisor, Department of Pathology, Edinburgh University; and J. C. Sommerville, A.I.M.L.T., Senior Technician, Department of Neuropathology, Edinburgh University. Foreword by Professor A. Murray Drennan, M.D., F.R.C.P.E., F.R.S.E. 10×7½ in. Pp. xi+199, with 262 figures, 30 in colour. Index. 1949. Edinburgh: E. & S. Livingstone Ltd. Price 35/-.

In recent years the Edinburgh School of Medicine, ably assisted by the publishing house of E. & S. Livingstone, has distinguished itself by the publication of a number of atlases in which the choice of subject and the execution of the illustrations are noteworthy. The present volume is a worthy addition to the series. As Professor Drennan writes in the foreword, it represents the "combined effort of the pathologist, who identifies and selects the lesion, the technician who makes possible its detailed study and the artist who perpetuates it for all to see." As in research, advance in teaching methods is thus fostered.

Fig. 43

Cerebral artery showing a mycotic aneurysm. The organisms in the infected embolus can be seen as a bluish haze at the periphery of the embolus. The arterial wall is weakened and bulging (upwards and to the right) and there has evidently been some leakage of blood (haemalum and eosin; ×80).

Fig. 54

Cerebral cortex in a case of meningococcal leptomeningitis. The surface is very congested and the creamy coloured purulent exudate is most abundant in the regions where the subarachnoid space is large, *e.g.*, along the sulci (as shown), in the basal cisterns and around the cauda equina. A large vein runs across the field.

The authors begin with illustrations of the normal histological structure of the central nervous system, and in a series of ten chapters progress through Vascular Disease, Infections, Demyelinisations, Intoxications, Degenerations, Trauma, Hydrocephalus, Tumours and Errors in Development. "The purpose of the atlas is to try to present to clinicians or pathologists beginning the study of neuropathology the most important pathological conditions in a clear and simple way." Towards this end the authors have been uniformly successful. In each of the chapters there are illustrations of the common pathological conditions. The naked eye lesion, as the student or pathologist sees it, is illustrated first. Then, in a series of well-chosen and competently executed photographs, the general features of the pathological process are made clear. Each illustration is accompanied by a descriptive legend, and in addition there is sufficient matter in the text to assist the beginner to an understanding of the disease. In many instances the illustrations are accompanied by a brief clinical history of the patient from whom the pathological specimen was obtained.

It is obvious that the value of such an atlas depends upon the choice of subject material and the quality of the illustrations. The authors have chosen wisely. The quality of the illustrations is in general excellent. A few of those in colour are not quite faithfully reproduced. The occasional diagram and drawing still further help the student to obtain a clear picture of the disease process. This atlas will be a useful companion to the young pathologist embarking on the study of neuropathology. To the clinician who takes an interest in the disease process it will be a valuable visual aid. Authors and publishers are to be congratulated upon the production of a book which is not one to borrow but to buy.—J. Henry Biggart.

DEMONSTRATIONS OF PHYSICAL SIGNS IN CLINICAL SURGERY. By Hamilton BAILEY, F.R.C.S. (Eng.), F.A.C.S., F.I.C.S., F.R.S.E., Surgeon, and Surgeon-in-charge of the Genito-urinary Department, Royal Northern Hospital, London; Senior Surgeon, St Vincent's Clinic and the Italian Hospital; Surgeon, Consolation Hospital, Lambeth; General Surgeon, Metropolitan Ear, Nose, and Throat Hospital, London. Eleventh edition. In four parts, Parts II, III and IV. $8\frac{1}{2} \times 5\frac{1}{2}$ in. Pp. 101–426, with 657 figures, many in colour. Index. 1948. Bristol: John Wright & Sons, Ltd. Price 8/6 each part. Paper cover.

In August 1948 we reviewed the first part of " Demonstrations of Physical Signs in Clinical Surgery " and now the remaining three parts have been published, of which the fourth is of most interest to orthopaedic surgeons in so far as it includes examination of the major joints, the peripheral nerves and the peripheral blood-vessels. The succinct text and the many telling illustrations are noteworthy. There is probably no surgeon who would fail to benefit from reading this work. For the postgraduate student this book is particularly helpful. He might perhaps find an easier discipline in a more systematic approach, particularly, for example, in the examination of joints. This is certainly true of the undergraduate, who would be sadly misled by such a statement as " mensuration is the first step in the examination " of the hip.

There are some shortcomings in the clinical pictures. In a description of sciatica, physical signs in the lumbar region are omitted altogether; in considering derangements of the knee joint a torn medial ligament is described as common, whereas a strain is not mentioned; calcaneal spur is regarded as a source of pain distinct from plantar fasciitis, and with a different site of tenderness; neurosis is implied to be the only cause of glove or stocking anaesthesia; and there are further examples. On the other hand, we welcome the common sense of such quotations as, " No orthopaedic surgeon draws Bryant's triangle. It suffices to kneel, facing the patient, and to put the thumbs on his anterior superior iliac spines, the middle fingers on the tips of the great trochanters and the ring and little fingers behind the great trochanters. Slight differences in the height of the trochanters are detected easily in this rapid way " (Alan Todd). We applaud the measurement of shortening by making the patient stand on wood blocks instead of making estimates with a tape-measure. The authors of signs and symptoms are quoted freely, and invariably their names are given in footnotes with an account of their principal appointments, together with the dates of birth and death of those no longer living. It would be even better if each name could be followed by a reference to the classical description of the eponymous observation.—H. Jackson BURROWS.

PHYSICS AND THE SURGEON. By H. S. SOUTTAR, D.M., M.Ch. (Oxon.), F.R.C.S. (Eng.), Hon. M.D., Trinity College, Dublin; Hon. F.R.A.C.S. Consulting Surgeon, London Hospital. $8\frac{1}{2} \times 5\frac{1}{2}$ in. Pp. vii+60, with 41 figures. 1948. Oxford: Blackwell Scientific Publications, Ltd. Price 7/6. (This volume will be No. 48 of the American Lecture Series.)

This neat slim book is full of interest though its few pages cannot hope to cover a very wide field, especially when over half are devoted to atomic physics. The mechanical principles of perennial interest to orthopaedic surgeons are illustrated by a number of line drawings, several of which, however, are hardly worthy of the text. For instance, in one figure the terminal phalanx appears to fuse with the nail, while the portly outlines of an oarsman are unflattering to British sportsmen, and make one inclined to doubt the magnitude of the forces developed in rowing. Abduction of the arm is described as taking place entirely at the shoulder till the right angle is reached, whereas it is now generally recognised that there is a gradual transition to scapulo-thoracic movement almost throughout this range. The account of atomic physics is clear and fascinating.—K. I. NISSEN.

ANATOMY AND BALLET. By Celia Sparger, M.C.S.P., C.C.P.E., Consulting Physiotherapist to the Sadlers Wells School. Foreword by S. L. Higgs, F.R.C.S., Orthopaedic Surgeon, St. Bartholomew's Hospital. Introduction by Ninette de Valois, C.B.E. $9\frac{3}{4} \times 7\frac{1}{4}$ in. Pp. 77. with 43 figures. 1949. London: A. & C. Black, Ltd. Price 12/6.

This book on Anatomy is written for teachers of Ballet. The orthopaedic surgeon will be surprised to read that the function of the infrapatellar fat pads is to prevent friction between the patella and tibia. But he will find much to interest him in the radiographs of hip and ankle joints taken in various dancing positions, and in considering the relationship to ballet of hallux valgus, internal derangement of the knee-joint and postural deformities of the spine.—R. WATSON-JONES.